Suid-Afrikaanse Tydskrif vir Geneeskunde
South African Medical Journal

Posbus 643, Kaapstad P.O. Box 643, Cape Town

Kaapstad, 19 Oktober 1957 Weekliks 2s. 6d. | Vol. 31 No. 42 | Cape Town, 19 October 1957 Weekly 2s. 6d.

CONGENITAL LYMPHOBLASTIC LEUKAEMIA IN AN AFRICAN MALE

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On 23 April 1957 one of us (H.F.T.) was asked to see a male African baby who had been born some hours before. The delivery had been uneventful and the period of gestation was normal. The mother appeared to be fit in all respects. She had not been exposed to X-rays as far as was known.[1,2]

Examination of the child showed the presence of small, discrete petechiae on the face, subcutaneous ecchymoses ranging in size from 1-3 mm. in the palms and soles, and subcutaneous haematomata scattered diffusely over the trunk. The visible mucous membranes showed marked pallor, but no icterus and no pinguecula could be discerned. The face was oedematous but there was no sign of oedema elsewhere. No mongolism was noted.[3,4] The umbilical cord did not bleed once it had been ligated. Dyspnoea was marked but there was no associated cyanosis or engorgement of the neck veins. Slight diffuse abdominal distension was present but clinical examination did not reveal any enlargement of the liver or spleen. No free fluid was found in the peritoneal cavity. The mucous membranes were not ulcerated, the superficial lymph glands did not seem to be significantly enlarged, and the remaining systems were not remarkable. No stigmata of congenital syphilis were found. No congenital morphological defects were noted.[5] Blood for haematological investigations was taken from the child's foot. Bleeding was free and could only be controlled by continuous pressure and by raising the foot.

An intra-tibial blood transfusion was started and the child received about 80 c.c. of blood before it died. Other treatment that was given included Konakion, 1 ampoule by mouth, intramuscular crystalline penicillin, 50,000 units 6-hourly, and streptomycin, ·05 g. every 12 hours.

The child lived for about 26 hours after its birth. The following are the results of the special investigations which were carried out:

1. *Blood Grouping:* Group O Rhesus positive. (The mother was also Group O Rhesus positive, and her Kahn test was negative.) A Coombe's test was not done.

2. *Blood Count.* Haemoglobin 6·25 g. (40%). Leucocytes 40,300 per c.mm. [polymorphonuclear leucocytes 15%, metamyelocytes 5%, 'blast cells' (probably lymphoblasts) 63%, lymphocytes 16%, monocytes 1%.] In each count of 100 leucocytes 7 early normoblasts were noted. The red cells appeared microcytic. Marked anisopoikilocytosis and polychromasia was noted. Platelets appeared to be considerably reduced in number.

3. *Bleeding Time* (Duke's method) markedly increased (bleeding persisted for more than 30 minutes).

4. *Clotting Time* (Wright's capillary-tube method) 1 hour.

5. *Prothrombin Index* 23% (control 20 seconds, patient 89 seconds).

In consultation between us a tentative diagnosis of congenital leukaemia was made.

Autopsy was performed about 12 hours after death, when the following relevant findings were made: The previously noted petechiae, ecchymoses and haematomata were more marked and more extensive than before. There was no special superficial lymph-gland hypertrophy. The peritoneal cavity contained a large amount of blood, which had not coagulated. The *liver* was enlarged, weighing 192 g. Its surface was smooth, its consistency softer than usual, and its colour normal. Subcapsular petechiae and ecchymoses were noted. In the right lobe a tear 1 inch in length extended from the right inferior margin along the inferior surface and between the colic and the renal impressions. The same tear also extended onto the right lateral surface for a further 1 inch. In addition a tear of the left lobe extended from the anterior margin backwards for about 1 inch. The *spleen* was enlarged, weighing 23 g. Its colour and consistency were normal. In both *lungs* large multiple ecchymoses could be seen subjacent to the visceral pleura and also diffusely throughout the parenchyma. *Pericardial* petechiae were seen. The heart was otherwise normal. The pericardial sac contained a small amount of straw-coloured fluid. The *brain* was very pale and oedematous and the convolutions were very flat. A few scattered petechiae were noted in the brain substance. The immediate cause of death seemed to be rupture of the liver with resultant severe peritoneal haemorrhage, and it was thought that cerebral oedema might be a contributory cause. It was considered reasonably certain that the rupture of the liver had occurred during the process of birth. It was believed, however, that the primary underlying cause of the whole condition was congenital leukaemia, probably of a lymphoblastic type.

The following are the results of the investigations carried out by the South African Institute for Medical Research:

On 9 May Dr. H. I. Lurie reported as follows:

Sections of the specimens from the *liver*, *spleen*, *kidney*, and *bone marrow* all show the presence of diffuse infiltration by immature cells, the histological features of which suggest lymphoblasts. Section of the specimen of the *thymus* shows no significant

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pathological changes. Section of the specimen from the *lung* shows that the bronchioles and alveolar spaces are filled with blood. The histological features are those of a *neonatal lymphoblastic leukaemia.*

On 18 May Dr. H. B. W. Greig reported as follows:

Peripheral Blood Smear: The red cells show moderate anisocytosis and poikilocytosis. There is severe hypochromasia. Platelets are reduced in numbers (estimated count 50,000 per c.mm.) and are bizarre in morphology. There is a marked leucocytosis, with many abnormal cells present, mostly of the lymphocyte series. Most of the lymphocytes are immature and many are frank 'blasts'. Bizarre monocytoid forms and primitive reticulum cells are present. There are many normoblasts present.

Bone Marrow. This marrow sample shows almost complete replacement of the normal marrow elements by primitive lymphoid cells. There are round cells with deeply basophilic rather scanty cytoplasm, often vacuolated, and a reticular nucleus usually containing 2-3 nucleoli.

This marrow picture, taken in conjunction with the peripheral blood picture, indicates a diagnosis of acute leukaemia, almost certainly lymphoblastic in type.

DISCUSSION

We considered that the patient had congenital leukaemia for the following reasons. He showed widely spread cutaneous haemorrhagic lesions at birth and died about 26 hours later. The peripheral blood showed the presence of large numbers of lymphoblasts. A high percentage of lymphoblasts was also found in the sternal bone marrow. Although normoblasts were found in the peripheral blood, the picture was not that of an erythroblastosis foetalis. Moreover there was no incompatibility in the bloods of the child and its mother. A marked thrombocytopenia was present and both the bleeding and coagulation times were considerably prolonged. The liver, spleen, kidney and bone marrow were invaded by primitive cells which were considered to be lymphoblasts.

Certain critera have been laid down for the diagnosis of congenital leukaemia,[5,6] as follows: Symptoms should be present at birth or develop very shortly thereafter. The liver, spleen and lymph nodes should be enlarged. The leucocyte count should be high and primitive white cells should predominate. There should be nothing in the history suggestive of syphilis, erythroblastosis foetalis or icterus gravis. There should be tissue infiltration by primitive white cells into non-haemopoeitic organs as well as the liver, spleen and lymph nodes. Primitive white cells should be seen in both the peripheral blood and the bone marrow. The possibility that the blood changes may be due to syphilis or to erythroblastosis foetalis should be excluded.

Certain features about our case were unusual. The patient was an African; we have been unable to trace any reference in the literature to congenital leukaemia in Africans. The leukaemia was lymphoblastic in type; the literature stresses the predominance of the myelocytic forms in this rare disease.[5-8] Several reports have appeared in the literature relating to the coexistence of congenital leukaemia and mongolism;[3,4] no signs of associated congenital disease were found of the type described in the cases of Bernhard *et al.*[5] There was no history of any exposure of the patient or his mother to X-ray therapy; such exposure may be important in the production of congenital leukaemia and this possible aetiological factor is receiving increasing attention in medical literature.[1,2]

SUMMARY

A case is described of congenital lymphoblastic leukaemia in an African male. The criteria for making a diagnosis of congenital leukaemia are discussed. The rarity of the lymphoblastic form of congenital leukaemia is stressed.

We wish to thank the medical staff of the South African Institute for Medical Research for their help in the investigation of this patient. A special word of thanks is due to Prof. J. F. Murray and to Drs. H. I. Lurie and H. B. W. Greig.

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VAN DIE REDAKSIE

TOLERANSIETOETSE VIR GLUKOSE

Die toleransietoets vir glukose word veral gebruik om vas te stel tot watter mate die weefsels in staat is om méér as die vastende hoeveelheid glukose op te neem. Terselfdertyd is die werklike syfer van die vastende hoogte moontlik ook waardevol. Die gewone mondelikse toleransie-kurwe ná die inname van 50 of 100 g. glukose hang ook van die dermopname van glukose af. Weens hierdie feit is die toets waardevol by die diagnose en berekening van sindrome van gebrekkige absorpsie waar 'n plat toleransie-kurwe nie vinnige assimilasie beteken nie, maar wel vertraagde absorpsie. Hierdie feit beteken egter dat die toets minder waardevol is by die berekening van glukose-toleransie, aangesien 'n mens moet begin met die ongeregverdigde veronderstelling dat die absorpsie-spoed altyd dieselfde is. Om hierdie rede is die toets ietwat gewysig sodat die glukose binneaars ingespuit kan word;[1] die ware spoed waarteen die glukose dan uit die bloedsomloop verdwyn kan in hierdie geval bepaal word. Die verskeie faktore betrokke by die gevolglike bloed-glukosehoogtes sluit in die diffusie in al die buitesellige vloeistof-spasies, die volgehoue uitskeiding deur die niere solank die bloedgehalte bó die nierdrempel bly, en die spoed waarteen die weefsels die glukose opneem totdat die vashoogte weer bereik word. Gelukkig, en ietwat onverwags, blyk dit dat die daling in die glukosegehalte vir die eerste uur ná 'n binneaarse toediening 'n eenvoudige en verklarende kurwepatroon volg.

Maar dit is nie so maklik om die resultate van die binneaarse toleransietoets uit te druk nie. Die assimilasie van glukose (d.w.s. die dalingspoed van die bloedsuiker) kan beskou word as in verband staande met òf die totale heersende bloedsuikerhoogte, òf met die verskil tussen hierdie hoogte en die voorafgaande aanvanklike vasgehalte. Hierdie twee veronderstellings verskil baie in die praktyk en gee aanleiding tot twee moontlike indices van glukose-assimilasie. Die eerste druk die spoed van assimilasie uit as 'n persentasie van die totale bloedsuiker (totale indeks); die tweede bereken die spoed as 'n persentasie van die betreklike bloed-suikergehalte bó die vastende hoogte (stygingsindeks). Daar is sekere bewyse dat die stygingsindeks die weefselopname van glukose meer akkuraat vertolk.[2] Dit staan onafhanklik van die ladingsdosis en die vastende bloedsuiker-gehalte, terwyl die totale indeks beïnvloed word deur 'n variasie in hierdie faktore. Dit is verder bevind dat die glukose-toleransietoetse op pasiënte vóór en ná karbutamied (BZ55) 'n ooreenkoms wys tussen die uitslae van die mondelikse

EDITORIAL

GLUCOSE TOLERANCE TESTS

The glucose tolerance test is designed primarily to ascertain the ability of the tissues to assimilate glucose, presented to them in excess of the fasting level. At the same time the actual figure for the fasting level may also be of value. The usual oral tolerance curve after the ingestion of 50 or 100 g. of glucose depends also upon the intestinal absorption of glucose. This fact renders the test of value in the diagnosis or assessment of malabsorption syndromes, in which a flat tolerance curve does not indicate rapid assimilation, but delayed absorption. However, this point renders the test of less value in glucose tolerance estimations, since one must start with the unjustifiable assumption of uniform absorption. For this reason the test has been modified in that the glucose may be injected intravenously.[1] In this event one can measure the true rate at which it subsequently disappears from the blood stream. The several factors concerned in the resultant blood-glucose levels include the diffusion into all the extracellular fluid space, the continued excretion through the kidneys as long as the blood level remains above the renal threshold, and the rate of uptake by the tissues until the fasting level is reached. Rather luckily and perhaps unexpectedly the fall in glucose level after an intravenous load seems to fit a simple exponential curve for the first hour.

When it comes to the method of expressing results from the intravenous tolerance test, it is not so simple. The assimilation of glucose (i.e. rate of fall of blood sugar) can be taken as related either to the total prevailing blood-sugar level or to the difference between this and the preceding initial fasting value. These two assumptions differ markedly in practice and give rise to two possible indices of glucose assimilation. The first expresses the rate of assimilation as a percentage of the total blood sugar (total index); the second expresses the rate as a percentage of the relevant blood-sugar level above the fasting value (increment index). There is some evidence that the increment index gives a more accurate expression of the tissue uptake of glucose.[2] It is independent of the loading dose and the fasting blood-sugar level, while the total index is affected by variation in

en dié van die binneaarse toets wanneer die uitslae as stygingsindeks uitgedruk word, maar daar was verskille wanneer die uitslae (van die twee vorms van die toets) uitgedruk was as totale indeks. Dit is verder bevind dat karbutamied die vastende bloedsuiker verminder, maar dat dit egter baie min verskil maak aan die mondelikse glukose-toleransie, bereken as die styging bó die vastende gehalte en die dalingspoed terug na die vastende gehalte. Dit maak dan ook geen verskil aan die binneaarse stygingsindeks nie, maar die totale indeks wys 'n merkbare vermeerdering in glukose-assimilasie onder die invloed van karbutamied.

Die binneaarse toleransietoets vir glukose kan aansluit by gepaardgaande insulien,[3] en lewer dus 'n direkte berekening van insulien-gevoeligheid of -weerstand, veral in verskillende groepe van diabetiese pasiënte. Hoewel dit eerder 'n metode van navorsing dan 'n prakties belangrike toets is, is dit ongetwyfeld 'n ondersoek wat baie meer akkuraat is as die mondelikse glukose-toleransietoets tesame met insulien.

Die grootste waarde van die binneaarse toets berus daarop dat dit die faktor van dermopname en die noodsaaklikheid om groot hoeveelhede glukose te sluk uitskakel; die feit dat die hele toets binne 'n uur afgehandel is; die toeretiese presiesheid van die uitslae wat behaal word; en die moontlikheid dat die toets met insulien verbind kan word. Aan die ander kant is dit by die mondelikse toets onnodig om 'n aarinspuiting te doen; geen aartrombose kan ontstaan nie, en die mondelikse toets sal dié pasiënte aandui waar die glikosurie veroorsaak word deur 'n vinnige absorpsie van glukose (die sg. ‚lag'-kurwe). Hoewel die toets toereties nie so betroubaar (as die binneaarse een) is nie, is dit nogtans baie waardevol in die algemene geneeskunde en selfs as 'n navorsingsmetode. Die kurwes wat behaal word is merkwaardig konstant, mits die toets onder vasgestelde kondisies gemaak word. Die ondervinding leer dat dit vir die gewone doeleindes nie nodig is om dit te laat vaar ten gunste van die binneaarse metode nie.

Die voorbereiding op die mondelikse toleransietoets is belangrik. Die pasiënt moet ten minste 4 dae lank voor die toets 'n volle dieet hou met ten minste 300 g. koolhidraat daagliks. Hy moet in vastende toestand wees met geen voedsel 12 uur voor die toets nie, en die 50 g. glukose moet in 200-250 ml. water opgelos word. Swart tee of koffie, sonder suiker, mag vir ontbyt geneem word. Die halfuurlikse bloedmonsters ná inname van suiker kan òf uit 'n aar òf uit die vinger geneem word. Die kapillêre monsters is miskien beter, mits hulle deskundig gedoen word, en hulle lewer glukosegehaltes wat ietwat hoër is as dié van aarbloed (behalwe by 'n vastende pasiënt). Monsters uit die haarvate is besonder nuttig om pasiënte uit te sonder by wie die bloedsuiker vinnig styg binne 'n halfuur, om op daardie tydstip glikosurie te veroorsaak, en dan tot die normale (of onder-normale) daal. Hierdie (‚steeple', ‚lag', of ‚oxyhyperglycaemic') kurwe kom voor na maagderminmonding of gedeeltelike maaguitsnyding, en ook by sommige lyers aan ‚funksionele hipoglikemie'.

Die glukose-toleransietoets is waardevol by die diagnose van ligte diabetes. As 'n vastende bloedsuiker van 200 mg. byvoorbeeld gevind word by 'n pasiënt met glikosurie, is dit nutteloos om 'n toleransiekurwe te neem. Dit is natuurlik ook tydverspeling om 'n toleransietoets te doen terwyl 'n pasiënt op insulien-behandeling is of 'n streng dieet hou.

these. Further, glucose tolerance tests on patients before and after carbutamide (BZ 55) have shown correspondence between results of the oral and the intravenous test expressed as increment index, while the total index differed from these. Carbutamide was found to lower the fasting blood sugar, but to make very little difference to the oral glucose tolerance, considered as the rise above fasting value and speed of return to this value. Similarly it made no difference to the intravenous increment index, but the total index showed a distinct increase in glucose assimilation under the influence of carbutamide.

The intravenous glucose tolerance test can be combined with concomitant intravenous insulin,[3] so affording a direct measurement of insulin sensitivity or resistance, especially in different groups of diabetic subjects. This, while remaining a research tool rather than a test of practical importance, certainly allows more accurate investigation than the oral-glucose tolerance combined with insulin.

The greater value of the intravenous test, therefore, lies in the abolition of the factor of intestinal absorption and the need to swallow a lot of glucose, the fact that the test is all over in an hour, the theoretical exactitude of the results obtained, and the possibility of its combination with insulin. On the other hand, the oral test does not necessitate intravenous puncture, cannot produce venous thrombosis, and will pick out the patient whose glycosuria is caused by rapid absorption of glucose (the so-called 'lag' curve). Although theoretically less exact, nevertheless its value in general medicine, and even as a research tool, is still great, and the curves obtained are remarkably constant, provided the test is carried out under standardized conditions. Experience teaches us that it is not necessary, for usual purposes, to abandon its use in favour of the intravenous method.

The preparation for the oral tolerance test is important. The subject should have had a full diet, including at least 300 g. of carbohydrate a day, for at least 4 days before the test. He must be in the fasting state, with no food for 12 hours, and the 50 g. of glucose should be dissolved in 200-250 ml. of water. Black tea or coffee without sugar is allowed for breakfast. The half-hourly blood samples after ingestion of sugar may be taken from vein or by finger prick. The capillary samples are probably preferable, provided they are expertly taken, and they give glucose levels somewhat higher than the venous blood (except in the fasting state). Capillary blood samples are particularly valuable for picking out the patient whose blood sugar rises rapidly within half an hour, to produce glycosuria at that time, and then falls to normal (or subnormal) levels. This ('steeple', 'lag' or 'oxyhyperglycaemic') curve is found after gastro-enterostomy or partial gastrectomy, and in some people who suffer from 'functional hypoglycaemia'.

The glucose tolerance test is of value in the diagnosis of mild diabetes. If, for instance, a fasting blood sugar of 200 mg. is found in a patient with glycosuria, the perform-

Dit is moontlik behulpsaam by die uitkenning van die voordiabetiese toestand, maar dit moet baie versigtig vertolk word. Nieteenstaande al die werk wat reeds in hierdie verband gedoen is, is ons nog nie seker van die normale speling by jong kinders, swanger vroue, en ou mense nie. Soos reeds gesê, is die toetse nuttig by die diagnose van ‚funksionele hipoglikemie', wanneer die bloedmonsters elke uur geneem moet word tot 6 uur na die inname van glukose. Dit het minder waarde by die hipoglikemiese toestand wat deur eilandselgewasse veroorsaak word; die kurwe is dikwels abnormaal en soms selfs diabeties van aard. By hipopituitêre kondisies en by Addison se siekte is die kurwe plat en kan die vastende gehalte laag wees. 'n Diabetiese kurwe kom soms by die Cushing-siekte en phaeochromocytoma voor, en die toleransietoets word hier diagnosties gebruik. Die veranderings by maagdermsiektes is reeds bespreek. Ook by ander siektes kan die toleransiekurwe van die normale afwyk, maar is klinies minder behulpsaam. Die kurwe is normaal by pasiënte met nierglikosurie, en beklink die diagnose by hierdie gevalle.

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ance of a tolerance curve is a useless waste of time. It is of course also useless while a patient is on insulin or on a strict diet. It may be useful in the detection of prediabetes, but needs to be interpreted with care. Despite all the work that has been done on it, we are still not quite sure of the normal range in young children, pregnancy and old age. It is useful, as mentioned above, in the diagnosis of 'functional hypoglycaemia', when the blood samples should be continued hourly up to 6 hours after the ingestion of glucose. In the hypoglycaemic syndrome caused by islet-cell tumour it is of less value, though often abnormal and sometimes even of diabetic type. In hypopituitary states and in Addison's disease it is flat and the fasting level may be low. A diabetic curve may be found in Cushing's syndrome and phaeochromocytoma and it is used as a diagnostic test for these diseases. The changes in gastro-intestinal conditions have been mentioned. In other disorders the tolerance curve may also be deranged, but is of less clinical help. In subjects with renal glycosuria the curve is normal, and establishes the diagnosis.

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A CRITICAL REVIEW BY THE MINISTRY OF HEALTH OF ENGLAND AND WALES OF THE REPORT OF A THOUSAND MATERNAL DEATHS*

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Three hundred years ago, in 1660, from information supplied by Sir James Y. Simpson and E. V. Sieveking, the maternal mortality rate in London was about 1 in 40 births and one hundred years later only about half the figure. A century ago a distinct improvement had occurred, for only about 1 in 200 women died in childbirth in England and Wales, a rate in modern terminology of 5 per 1,000.

We are about to consider a survey of the causes of over 1,000 maternal deaths when the rate over a period of 3 years was slightly over one-tenth of that figure.

THE REPORT

The Report on a Confidential Enquiry on Maternal Deaths in England and Wales that was published in July this year contains information of the greatest interest and value. The investigation concerned was the second of its nature and followed a similar, if simpler, enquiry, namely that of a Departmental Committee on Maternal Mortality and Morbidity appointed in 1928 by the then Minister of Health. The Committee published a final Report in 1932 and, whereas many imperfections were apparent in the lack of, or method of, collection of necessary material, the results were considered fully to justify the continuation of the work. This was done year by year and the findings appeared in successive Annual Reports of the Chief Medical Officer of the Ministry of Health. In 1949 maternal mortality was a subject for discussion at the XIIth British Congress of Obstetrics and Gynaecology held in London. At the time reference was made to a parallel investigation into maternal deaths in the USA, in which there were apparent obvious advantages in the use of expert opinion. The President of the Congress, Sir Eardley Holland, suggested to the Minister of Health the possibility of adopting a similar method in England and Wales and, after consultation with various bodies, a new scheme was drawn up for the obtaining of information, which scheme was so designed as to include evidence from all who might be able to contribute to the explanation of any maternal death.

This improved method of enquiry came into operation in 1952 and, presented in the briefest manner possible, it consists in the collection by the medical officer of health of the district of statements from the local authority's domiciliary midwives or antenatal clinics, from hospitals or

* A paper submitted at the South African Medical Congress, Durban, September 1957.

general practitioners, or from anyone else who could possibly assist. In every instance the information is forwarded to the local consultant obstetrician for the area who, as a most valuable member of the team of enquiry, is able to add details known to himself or, when desirable, can obtain further additional and valuable clinical evidence. At this stage the enquiry form is nearing completion and is submitted to the Regional Assessor, who is a senior obstetrician of high standing in the area of each Regional Hospital Board. From the statements made on the report, amplified if desirable by further confidential enquiry, the regional assessor states his opinion about the cause of death and also whether any avoidable factor is present. The final assessment and classification of the reports is then done by the Ministry's consultant advisers in obstetrics, and it is here possible to attempt to iron out the slight variation in standard inevitably existing among the 15 regional assessors and thereby to present a more uniform picture.

The whole enquiry, which is anonymous, is regarded as strictly confidential and for medical use only. Apart from its value in the production of a report, all concerned have had an opportunity to review the clinical course of the patient and of the action taken. Each must benefit from this, however experienced he or she may be, or whether his or her work was performed in a hospital obstetric unit, a maternity home, or the patient's own home. As one whose task it has been to review every report, I can testify how greatly they contributed to my own knowledge of obstetrics. It is my intention to try to present in this address what appears to me to be some of the findings of major importance and interest, from which, therefore, the most valuable lessons may be learned.

The series covers not only deaths directly due to pregnancy and childbirth but also deaths ascribed to other conditions occurring in association with pregnancy and childbirth, as, for instance, heart disease. These will be referred to as 'associated' deaths. In all, confidential reports had been received of 1,094 deaths in the former group and 316 'associated' deaths during the 3 years 1952-54. The *International Statistical Classification of Diseases, Injuries and Causes of Death* (6th revision, 1948) was used and it is not proposed to consider the difficulties that were occasionally experienced in allotting the correct rubric.

Throughout the Report attempts were made to assess what was termed 'the primary avoidable factor'. Never, except when this factor pointed to deficient administration was this referred back to those in clinical charge of the patient. Its assessment was for one purpose, and one purpose only, and that was constructive and not destructive. The whole object in its determination was to see whether it would be possible, by discovering the nature of factors that contributed to the deaths of these women, to identify any that might be avoided in the future. If this was the case, then good would be done by their publication and the likelihood of their repetition probably lessened. It will be seen that in this respect much may be accomplished. It was to the regional assessors only that the direct questions were put: 'Do you consider that any avoidable factor existed and if so at what point did it lie?' The replies were either 'No' or 'Yes' or 'Doubtful'. The 'doubtfuls' were not numerous and were always transferred, usually because the evidence was not sufficient, into the 'No avoidable factor' group. As a final editor, however, I had no doubts whatever that in the great majority of cases labelled 'doubtful' an avoidable factor did exist and might have been discovered after further searching enquiry and personal questioning. This course was never followed as it would inevitably have destroyed mutual confidence and would soon, as a direct result, have destroyed that atmosphere of cooperation and willingness to help. In short, it would have done more harm than good.

The preliminaries may now be considered to be over and attention can be directed to the more important and clinical findings. In order that the whole subject be viewed in a correct perspective and lest the subsequent review of the causes of maternal death in childbirth convey an entirely erroneous and exaggerated impression of the very great safety of this desirable and physiological process, it is necessary to remind those who already are aware, and to tell those others who are unaware, of the fact that the figure for maternal mortality has sunk steadily over the last few decades to a level that would hardly have been thought possible 50, or indeed 25, years ago. This is immediately apparent when it is pointed out that the 1,410 deaths reviewed in this Report (representing some 80% of all maternal deaths) are but a tiny fraction of the 2,052,000 confinements that occurred in England and Wales during the 3 years 1952-54; 25 years ago a similar number of deaths would have occurred in 6 months, instead of 3 years. It was in spite of this tremendous improvement, and in the belief, however, that still more might be accomplished, that the present enquiry was initiated. Let us consider what it has achieved; in this account the mention of figures and statistics will be reduced to a minimum.

Primary Avoidable Factor

In the enquiry the first fact that emerged was that a primary avoidable factor existed in certainly no less than 43% of all deaths in the series. It is interesting to note that in a recent publication of a parallel enquiry in Massachusetts, Jewett[1] gave an almost identical figure of 47%. In both series about 10% of the cases were classed as 'doubtful' or 'undetermined', and for reasons already stated it would be fair to say that an avoidable factor was present in at least half of all maternal deaths.

Causes of Maternal Deaths

The nature of the chief causes of maternal deaths in England and Wales in the enquiry series can be set out as in Table I. These four conditions together account for two-thirds of the deaths due to pregnancy and childbirth.

TABLE I. CHIEF CAUSES OF MATERNAL DEATHS *

	No. of cases	*% of total*
Deaths due to toxaemia (including toxaemic antepartum haemorrhage)	246	22
Deaths due to haemorrhage (excluding toxaemic antepartum haemorrhage)	188	17
Deaths due to abortion	153	14
Deaths due to pulmonary embolus	138	13

* 1,094 deaths, in the 3 years 1952-54 directly due to pregnancy or childbirth (England and Wales).

In the 316 deaths associated with pregnancy and childbirth, heart disease is pre-eminent, accounting for no fewer than 121 cases or more than one-third of the total.

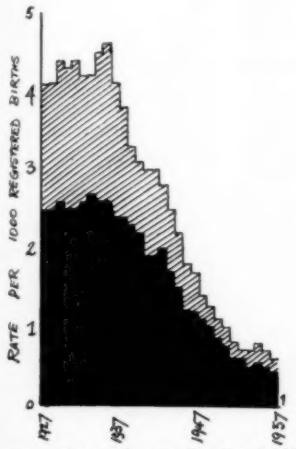


Fig. 1. Maternal mortality (per 1,000 births) from (1) sepsis and (2) other causes.

Attention is directed to the humble position occupied by puerperal sepsis. Not long ago this condition could truly be described as the nightmare of midwifery. Year after year it headed the list of fatal causes of maternal death and its position appeared impregnable. It was not affected by the efforts of clinicians or administrators to provide a better service, or by the greatly improved education of medical students and midwives, or by the provision of increased facilities for hospital confinements; the institution of antenatal clinics and the widespread appreciation of prenatal care made no difference and, lastly, no effect was apparent concurrent with the rapid advances of our general knowledge in all branches of medicine. In the present enquiry puerperal sepsis accounted for only 42 deaths or about 4% of the total deaths. In the series investigated by the Departmental Committee on Maternal Mortality and Morbidity whose report was published in 1932, the comparable figure for puerperal sepsis was 37% of maternal deaths. The steady and truly dramatic decline in the contribution made by puerperal sepsis is shown in Fig. 1.

TOXAEMIA OF PREGNANCY

Toxaemia of pregnancy now occupies the unenviable position as the chief cause of maternal death; in all, 246 cases, or nearly one-quarter of the deaths due to pregnancy and childbirth in this series. Avoidable factors were believed to be present in 128 cases or 52%.

The 246 cases were divided on clinical evidence into 'pre-eclampsia', 'eclampsia', and 'accidental haemorrhage with toxaemia', and it is of interest to note that no fewer than 90 fell into the first group (pre-eclampsia). This fact appeared to be unusual in that death occurring in the pre-eclamptic state was not commonly observed. However, the reports contained a full account of the post-mortem findings in 68 instances (Table II) and this left only 22 in which the pathologist's opinion was inconclusive.

For some years there has existed a general appreciation of the increased risk of death from toxaemia with increasing age and increasing parity. The present investigation clearly confirms this fact, as is demonstrated in Figs. 2 and 3. In a break-down of the figures according to the presence or absence of an avoidable factor no consistent variation was apparent in the matter of the parity, but this did not seem to be so in the case of the age of the patient. Reference is made below to a possible explanation (in the review of the nature of the avoidable factor).

TABLE II. POST-MORTEM FINDINGS IN 68 DEATHS FROM TOXAEMIA OF PREGNANCY

Massive hepatic and/or renal necrosis (including acute yellow atrophy of the liver)	29
Cerebral haemorrhage	13
Cardiac failure (severe hypertension)	9
Uraemia	5
Cerebral thrombosis	4
Pulmonary embolus	3
Renal cortical necrosis	2
Peritonitis after Caesarean section	2
Suprarenal haemorrhage	1
	68

A survey of the arrangements made by the patient for her confinement brings out information of interest and perhaps of importance and is summarized in the following

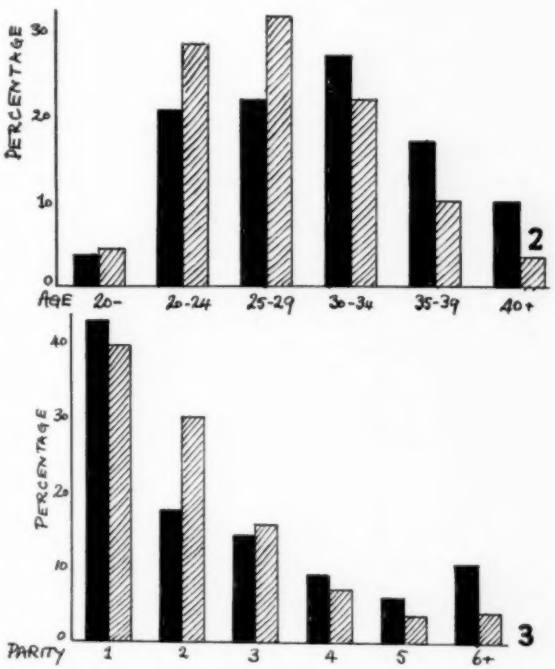


Fig. 2. Percentage distribution of maternal deaths from toxaemia (shown in black) and of all registered births (shown hatched) according to maternal age.

Fig. 3. Percentage distribution of maternal deaths from toxaemia (shown in black) and of all registered births (shown hatched) according to parity.

Fig. 4. It is at once clear that there is a great difference in the proportion of deaths with avoidable factors amongst those women who had made arrangements for their confinement at home compared with those who had placed themselves under the supervision of a hospital. There were 98 women in the first category and in 62 of these, or 63%, avoidable factors were considered to be present, whereas of the 83 who had booked at hospital an avoidable factor was obviously present in only 22, or 26·5%.

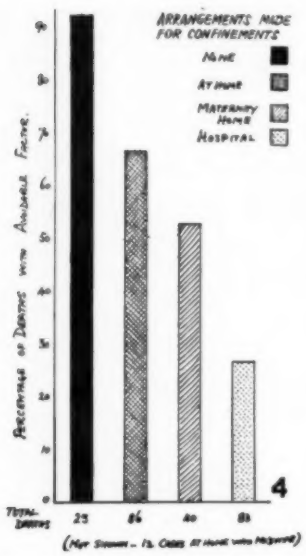


Fig. 4. Toxaemia: percentage of deaths with avoidable factor according to arrangements made for confinement.

It is necessary to add at once that these figures must not be taken to indicate that only the antenatal care received in hospital is satisfactory. Such indeed was not found to be the case; the figures may indicate that the appearance of toxaemia of pregnancy is an indication for hospital care and that this suggestion was not implemented.

It is only by a scrutiny of the nature of the avoidable factors that we may expect to learn where the obstetric service is likely to fail and from this metaphorically to make an attempt to plug the holes, however small they may be, in a leaking ship. In many cases more than one avoidable factor was present, but of all perhaps the most striking (to me at any rate) was the part played by the patient herself. The report shows that in a number of instances, in spite of the fact that the woman was really ill and in spite of the urgent requests of doctors and midwives, the suggestion that arrangements be made for her immediate admission to hospital was turned down time and again. In this the sick patient was often supported by ignorant or stupid relatives. Whether more can be done by health and education services in the future remains to be seen.

The most serious contributory cause to the group with an avoidable factor lay in the lack of provision for adequate antenatal care. On 59 occasions it was apparent that there had been an obvious failure in the accepted standard of prenatal supervision. Again it is only fair to point out what a minute fraction this constitutes of the total antenatal visits of the two million women who underwent their confinement in the years under review. The figure may indeed be increased by those forms registered as 'doubtful', but this small addition would make no appreciable difference. Nevertheless, antenatal care did fail and need not have failed—and this both in quality and quantity. Faults were found in all branches of the service. If the patient's arrangements were made for confinement at home transfer to hospital was delayed until it was too late; or if she were under the care of a hospital her admission on the appearance of symptoms and signs of toxaemia was not immediate. An excuse, for which there existed no justification whatever, was occasionally made that no beds were available. If genuine difficulty did indeed exist, it indicated the necessity for an immediate survey of bed allocation by both clinicians and administrators, who should be guided by the fact that the number of antenatal beds in any obstetric unit should be assessed, not in proportion to the number of beds provided for parturient women, but rather on the size of the population served by the hospital.

Another fault in the antenatal service repeatedly appeared. Either no record was kept by the hospital, clinic, doctor or midwife of the dates on which the patients were to be next seen, or if such a record were kept and the patient did not attend, no effective steps were taken to discover why. Time and time again the reason for non-attendance was discovered, too late, to be illness of the patient. The value of an accurate attendance-card system and the rigid adherence to the rule that non-attendance demands immediate enquiry—and this not by letter post—must surely be accepted. In the present series in every case but one this particular failure was recorded in the antenatal service attached to a hospital unit.

In a number of instances the avoidable factor lay in the arrangement for the confinement to take place at the patient's home. The mistake was indisputable in this group and it would not be unfair to state that it was the cause of death in these women. Thus home confinement was agreed to when the woman had had pre-eclamptic toxaemia in previous pregnancies, or when she was 40 or more years of age.

HAEMORRHAGE

In all, no fewer than 220 women in the series under review lost their lives from loss of blood. Haemorrhage was either the direct cause of death or it initiated a train of events that led to death, as in some cases of accidental haemorrhage in which the patient ultimately succumbed to anuria.

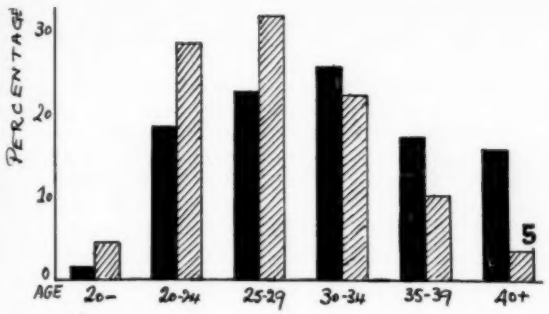
The 220 cases were grouped as shown in Table III. All

TABLE III. DEATHS DUE TO HAEMORRHAGE

	Cases
Accidental haemorrhage	78
Placenta praevia	29
Postpartum haemorrhage with retained placenta	53
Other postpartum haemorrhage (excluding haemorrhage following Caesarean section)	60

the deaths from accidental haemorrhage are included, whether they were thought to be due to pre-eclamptic toxaemia or not. This was done because, whatever their cause, points in common were present in their management. There is also the well-known difficulty in the clear demarcation of toxaemic and non-toxaemic accidental haemorrhage. Deaths due to haemorrhage following Caesarean section have been excluded, since these were regarded clinically as a post-operative complication; they are considered below.

The occurrence of death from haemorrhage in relation to age and parity are shown in Figs. 5 and 6. Again they confirm clinical experience and show that the risk of haemor-



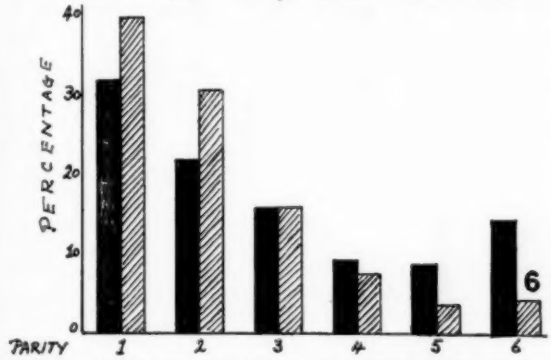


Fig. 5. Percentage distribution of maternal deaths from haemorrhage (shown in black) and of all registered births (shown hatched) according to maternal age.

Fig. 6. Percentage distribution of maternal deaths from haemorrhage (shown in black) and of all registered births (shown hatched) according to parity.

rhage is greater in the older age-groups and in women of the higher parity.

It is when attention is drawn to the incidence of the avoidable factor that valuable information may be forthcoming. Figs. 7 and 8 show the proportion of deaths with avoidable factors present in relation to the arrangements made for the confinement. Allowance must, of course, be made for the small number of cases under a few of the headings, but it is clear that wherever the arrangements were made for the confinement to take place at the patient's own home or in a maternity home the incidence of avoidable factors was raised and was particularly high in cases of post-partum haemorrhage. In regard to the significant difference in the incidence of avoidable factors when arrangements were made for a domiciliary confinement as compared to the hospital series, the words of the Report read: 'This, of course, is not to say that domiciliary care is less safe for the normal, but it does suggest a greater risk, as might be expected, for the abnormal or for those cases in which, by reason of higher age or parity, there is likelihood that abnormality may arise.'

The analysis of the avoidable factors falls in the main into the headings considered already under toxaemia of pregnancy. Thus it was found that the quality of antenatal supervision was inadequate, in that no notice had been taken by the patient or by the doctor of slight, but warning, loss of blood in pregnancy, or that antenatal care was irregular, or that difficulties were said to be experienced in the arrangement for admission to hospital or, lastly, that the arrangement for domiciliary confinement was made in most unsuitable cases. But it is in the management of the actual haemorrhage when it had occurred that the majority of avoidable factors were discovered. It is necessary here to remind you that in the small and thickly populated British Isles, the practical value of the Emergency Obstetrical Team, usually called the Flying Squad, has over recent years been proved time and again. These teams are so organized and so placed that they are within reach of about 30 minutes of any but the very few thinly populated parts of the country. At any rate, this applies to the whole of England and Wales and to most of Scotland. It was the lack of appreciation of the value of summoning expert aid to the patient that made all the difference between life and death. Instead of doing so, arrangements were made for the transfer of the patient, often only a very short distance, to hospital, and this when she was either dangerously ill through loss of blood, or alternatively when it was probable that the disturbance that must inevitably accompany the journey would almost certainly precipitate further, and perhaps fatal, loss of blood in a case of placenta praevia. The Flying Squad possesses the personnel, skill and equipment simply and safely to resuscitate the patient and has thereby almost removed the hazards of the subsequent transfer of the patient from her home to the hospital.

Another point that became apparent was the lack of appreciation of the risk of moving a patient from her home to a hospital with the placenta undelivered. In at least 21 instances this provided a primary avoidable factor. In many of these fatal cases comparatively little haemorrhage had occurred before arrangements were made for the trans-

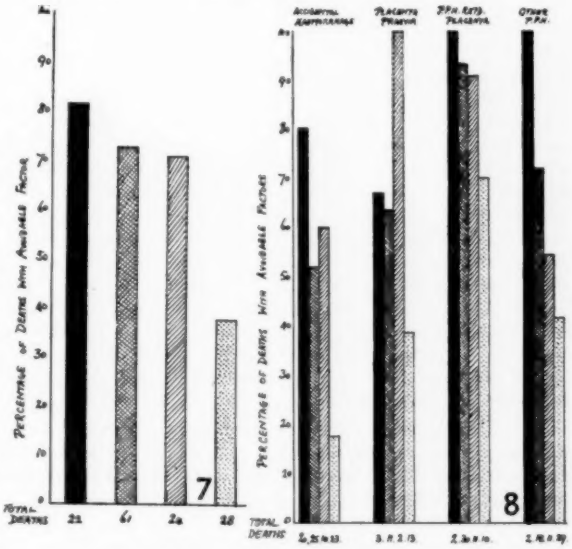


Fig. 7. Haemorrhage: percentage of deaths with avoidable factor according to arrangements made for confinement (arrangements indicated in same manner as in Fig. 4).

Fig. 8. Accidental haemorrhage, placenta praevia, PPH with retained placenta, other PPH: percentage of deaths with avoidable factor according to arrangements made for confinement (arrangements indicated in same manner as in Fig. 4).

ference of the patient, and in every case she arrived almost moribund and in a state of shock that proved irreversible.

There is no doubt that the move to hospital of women who are still in the third stage of labour has been done in the past and still is done today. The doctor should ask himself most seriously whether alternative arrangements would not be wiser.

PULMONARY EMBOLISM

Although it was found that the cause of death in 138 women was massive pulmonary embolus, it is perhaps sad to have to admit that, partly owing to lack of information, but mostly owing to our almost complete lack of understanding of this condition, no constructive information was obtainable. The incidence of avoidable factors was therefore considered negligible.

ABORTION

In the series under review death from and directly due to abortion occurred in no fewer than 153 women, and this condition occupies fourth place as a cause of maternal mortality. In 108 instances the death was regarded as avoidable because the abortion was known to have been procured by the woman herself in 58 instances and by some other person in 19 instances, and in the remainder the specific information was lacking. A few facts of importance emerged from the reports. Firstly, the number of procured abortions was just 4 times as great in married as it was in single women. It was further noted that the majority of single women were living in what was classed as 'comfortable' circumstances, whereas a considerable number of the married women were classed as 'poor'. This suggests that the latter group, living under poor conditions, might have had a struggle to keep a home together and acted in the belief that they could not contemplate another addition to the family. Sepsis accounted for over half the deaths and air embolus for a quarter. In 50 instances of procured abortion the method used was the injection under pressure of some fluid, usually soap and water, into the vagina and occasionally, possibly, into the cervix. Every single death from air embolus followed the use of this method. Bacteriological examination showed the dominance of *Cl. welchii* as the pathogenic organism in those cases where infection was a factor. All with experience of the care of patients who have procured abortion, or have had abortion procured, will already know that for every death countless other women go through a dangerous illness from which they may never completely recover. Perhaps a minor complication is that many of them are left sterile.

The problem is just as much a social problem as it is medical, but it is one that will have to be tackled sooner or later all over the world.

CARDIAC DISEASE

Following closely in importance on abortion, cardiac disease associated with pregnancy and childbirth accounted for 121 deaths.

The age analysis (Fig. 9) again shows the increased risk amongst the older age groups. Fig. 10, however, tells us that death from cardiac disease, in the great majority of cases mitral valve disease, occurs in a high proportion of first pregnancies. There seemed to be no special time in pregnancy when death was likely to occur in this group and the incidence here of what was considered to be an avoidable factor was low. The explanation lies probably in the very great difficulty in giving an accurate prognosis of the manner in which a diseased heart is likely to behave during a first

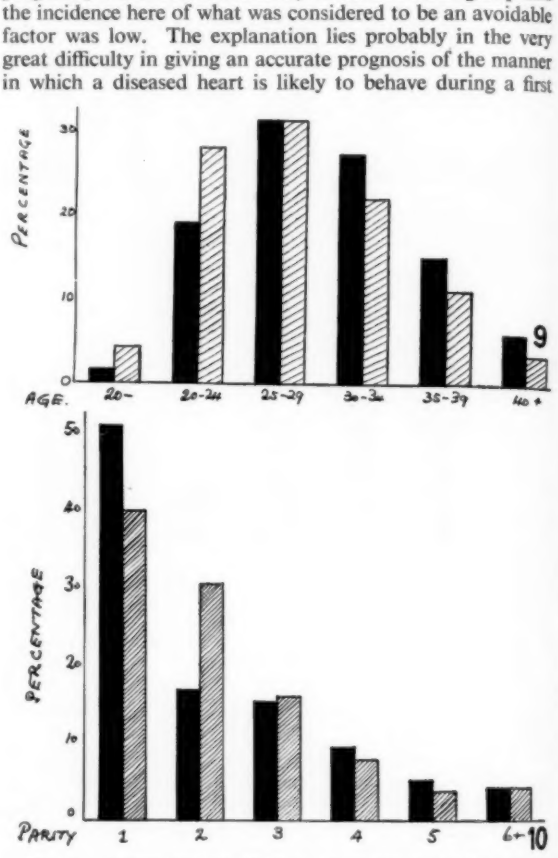


Fig. 9. Percentage distribution of maternal deaths from cardiac disease (shown in black) and of all registered births (shown hatched) according to maternal age.
Fig. 10. Percentage distribution of maternal deaths from cardiac disease (shown in black) and of all registered births (shown hatched) according to parity.

pregnancy. About one-third of the cases died in pregnancy, about one-third died during or immediately after delivery, and the remaining third during the puerperium. This is mentioned because it is commonly taught that death from heart failure is not common during labour and that the most dangerous time for the mother is in the early days of the puerperium.

An analysis of the incidence of the avoidable factor once more shows a repetition of findings that have already been noted. An avoidable factor was obviously present in about one-third of the total number of cases in which death occurred from heart failure, and this proportion would be appreciably increased by a number of records that from lack of necessary information had to be classed as 'doubtful'.

Once more the patient herself, aided by ignorant and probably stupid or selfish relatives, provided an avoidable

factor in that the acceptance of advice given about the wise management of pregnancy was flatly refused. In a few instances antenatal care, for one reason or another, was frankly inadequate, but throughout the series there existed a definite, and what might be termed general, failure to appreciate the absolute value of, and necessity for, a reasonable period of rest in bed in hospital for some weeks before the onset of labour.

Several of the patients were multiparae and in these cases the assumption appeared to have been made that because the woman had successfully accomplished previous pregnancy, labour and the puerperium with little evidence of cardiac embarrassment, therefore this happy state could be anticipated indefinitely. It was as though the problem was regarded as being analagous to that of the patient who was suspected of having a slight degree of pelvic contraction and whose first confinement provided valuable information on the likelihood of normal labour in the future. Such an analogy, of course, is completely unjustified in any woman suffering from valvular disease of the heart, for this surely is a progressive disorder. That such indeed was the case was demonstrated in those women whose death occurred from heart failure in their 5th or 6th pregnancy.

Occasionally, although a cardiac abnormality was noted, no attempt was made to obtain expert advice from a suitably experienced physician. Sometimes arrangements were made for these patients to be confined in their own homes—certainly a most unwise proceeding, for it is surely impossible accurately to give a prognosis about cardiac function in pregnancy or labour.

The advantage of a hospital booking for these women is clear. It ensures a hospital bed in the event of early symptoms of impending trouble; it ensures a hospital bed for a routine period of complete rest in the pregnancy; it facilitates the availability of expert advice and, lastly, in the event of heart failure, life-saving treatment is likely to be much more efficient aud more readily provided.

CAESAREAN SECTION

The fact that death followed delivery by Caesarean section on no fewer than 183 occasions out of the total of 1,410 records received during the period under review indicated that a careful scrutiny of these cases was merited and was likely to be productive of information of importance.

In the first place, by a somewhat complicated, but probably accurate, method of calculation it was possible to estimate that the incidence of Caesarean section in England and Wales during the 3 years 1952–54 was approximately 28 per 1,000 of all deliveries; also that the mortality rate for these cases was at the rate of 4 per 1,000, which may be compared with the total maternal mortality rate (excluding abortion) of 0·6 per 1,000 cases. This agrees very well with figures given by Marshall and Cox[2] (1949). In a total of over 7,000 Caesarean operations performed between 1943 and 1947 a maternal mortality figure of 9·9 per 1,000 was discovered. If, however, an estimate was made in those cases in their series in which section was premeditated and the general state of the patient was good, for 850 upper-segment operations the mortality rate was 3·5 per 1,000 and for 2,276 lower-segment operations only 1·3 per 1,000.

The figure of 4 per 1,000, calculated in the Report, is of interest as it indicates the risks, or safety, of the procedure in a series of cases on a national basis as distinct from a series presented by any one individual surgeon or occurring in any one obstetric centre.

Before the causes of death are considered it is necessary to state that although the deaths followed Caesarean section the fatality was by no means always directly related to the operation. Table IV shows the incidence of what was

TABLE IV. CAUSE OF DEATH IN DEATHS FOLLOWING CAESAREAN SECTION

Immediate Cause of Death	*No. of cases*	%
Haemorrhage	35	20·0
Pulmonary embolism	32	18·3
Shock or Collapse	30	17·1
Sepsis	14	8·0
Ileus	12	6·9
Asphyxia from inhalation of vomit during anaesthesia	12	6·9
Nephritis	7	4·0
Cardiac failure	6	3·4
Pneumonia	5	2·9
Eclampsia	5	2·9
Other causes	17	9·7
Total	175	100·0

believed to be the cause of death, and subsequent reference is made only to the 6 conditions that head the list. As the report states, it is surprising to see that haemorrhage was the chief cause of the loss of the mother's life. The true figure may well be considerably higher, for excessive loss of blood must influence the likelihood of pulmonary embolism and again from the clinical observations made in the records haemorrhage almost certainly preceded many of those included in the heading 'shock or collapse'. Marshall and Cox[2] in 1949 recorded an exactly similar observation. In their series of cases half the deaths following Caesarean section occurred within the first 3 days and the cause was noted to be haemorrhage and shock in the great majority.

The conditions of haemorrhage, pulmonary embolism and shock or collapse resolve themselves into one group, for in the majority—48 out of a total of 65 or 73·8%—the operation was premeditated and the patient was not in labour. In the second group, sepsis and ileus, out of 26 cases exactly the reverse was seen, for the patient had been in labour, sometimes many hours, in 18 cases or 69·2%. It is obvious, therefore, that a different set of circumstances were likely to be present in each group, and such indeed is what was found. In the smaller group, where death was certified as being due to sepsis and ileus following an operation performed usually after the patient had been in labour or the membranes had been ruptured for many hours, the sequence of events requires no clarification. It is to the first group, where death occurred directly or indirectly as the result of loss of blood that attention is directed, for surely in these days this occurrence must be regarded as unusual, if not unnecessary, in hospital practice. Arrangements for Caesarean section, when premeditated, should only be made in a suitable hospital and this was not always done. Many contributory factors were found to be responsible and often more than one was present. Thus it appeared that frequently insufficient attention was paid to the general condition of the patient immediately before or at the time of the operation. Extending over the preceding weeks a

state of anaemia had received inadequate treatment. Alternatively, there had been a lack of appreciation of the cumulative effect of repeated if apparently small losses of blood, or of the dangerous possibilities of a subsequent and more severe haemorrhage. Patients were despatched by ambulance whose condition was poor at the start of the journey and perhaps desperate on arrival at hospital or, in other words, inadequate consideration was given to the advisability of instituting steps directed towards resuscitation in the home by the emergency obstetric team. It is never possible at the time of operation to foretell the amount of blood that will be lost, but when Caesarean section is performed for placenta praevia, and if at the time there are found to be present in the lower segment of the uterus a collection of huge blood vessels, the wisdom of making the incision through the uterine wall for a lower-segment operation as a routine, and therefore through an obviously highly vascular and dangerous area, seems at least to merit some thought. In several instances in this series death occurred in just this manner from truly uncontrollable haemorrhage, which might have been avoided by the siting of the uterine incision at a slightly higher level and away from certain danger. Lastly, haemorrhage was seen to occur after completion of the operation, perhaps after the surgeon had left the hospital. Delay occurred in some cases in the initiation of rapid blood transfusion and this was due either to inadequate preparation in the preliminary estimation of the patient's blood-group and the provision before operation of suitable blood, should transfusion be necessary, or occasionally to the performance of the operation in most unsuitable maternity homes or small hospitals, both of which would lack the necessary expert and technical assistance.

DEATHS DUE TO THE COMPLICATIONS OF ANAESTHESIA

In the series under review death occurred from this cause in 49 cases and it is probable that in at least 20 others the anaesthetic was a contributory factor. The fact that in 32 instances death was caused by the inhalation of stomach contents appeared to merit attention, especially so as this complication must have occurred on very many other occasions when, while it was not fatal to the woman, the state of anoxia possibly killed the undelivered foetus. The indications for the anaesthetics in this series were varied, as also were the numerous anaesthetic agents used. Whenever an anaesthetic is required for a woman at the end of pregnancy, and especially toward the end of labour, the case is fraught with potential dangers. Both anaesthesia and childbirth in these days are such safe procedures that every consideration must be given to any preventable cause of disaster. The depth of anaesthesia needed is usually light, partly in order that at the subsequent delivery the living baby should respond quickly to the stimuli initiating respiration and partly to preserve the power of contraction and retraction of uterine muscle. The lightness of anaesthesia in itself increases the possibility of regurgitation of stomach contents and demands greater skill in its administration. The patient is not always in good condition, for she will be tired, may be exhausted, and may already have lost blood. In a praiseworthy attempt to prevent ketosis she will probably have been encouraged in labour to take frequent glucose drinks and the stomach is not likely to be empty. The presence of the large pregnant uterus itself tends to embarrass normal respiration and heart action, and this is not lessened by the use of the dorsal position and still less by the lithotomy position now generally adopted respectively for natural and forceps delivery. The dangers of the inhalation of regurgitated stomach contents are considerably lessened when the delivery is conducted with the patient lying on her left side. In the event of regurgitation it is of great assistance immediately to be able to tip the labour-ward table head downwards and the value of an efficient suction apparatus is undisputed. Neither of these will be available except in the labour ward of a hospital. It is strange to think that, whereas tremendous and life-saving advances have been made in nearly every other branch of anaesthetics over the last two or three decades, our colleagues have so far directed little attention to, indeed it would not be unfair to state that they have shown little interest in, the problems and dangers of the administration of anaesthetics to women in late pragnancy or in labour.

CONCLUSION

Has it all been worth the trouble? This question might well be asked—indeed, to my certain knowledge, it has been asked. With a national maternal mortality rate as low as 0·6 per 1,000 registered births is much more likely to be achieved? Remarks are added to the effect that there must be an irreducible minimum and that assuredly that figure is being approached. I clearly remember, however, that exactly the same things were believed and stated 30 years ago, when the maternal mortality in Britain was 6 times as great as it is today; and the Report which is being considered has demonstrated that even today in approximately half the maternal deaths there exists an avoidable factor. Childbirth is becoming more and more safe and, if we are prepared to study lessons from material such as has just been provided, and to act upon them, a maternal mortality of 2, or even 1, per 10,000 births can be expected. Indeed, the number of maternal deaths registered in England and Wales fell by a further 11% in 1956 as compared with 1955.

Lest I be accused of placing before you an unreasonable and fanciful suggestion, I can at least claim that I am not alone in my belief. In his recent article on maternal mortality in Massachusetts, John Figgis Jewett[1] writes as follows: 'With this background of the past and present, one should consider now the thesis that, no matter how creditable the rate, it can always be reduced. There is a tendency to leave the proper fields of obstetrics and to dabble in others, deluded by the false assertion that an "irreducible minimum of maternal mortality has been achieved".' Jewitt goes on to point out that foetal perinatal mortality is inevitably linked with maternal mortality and that as long as the perinatal foetal mortality rate is in the region of 30–40 per 1,000 live births it is, to use his own words, 'incredible that anyone would suggest such a threat to survival as all but ended'.

The immense amount of work that has been necessary for the completion of this report—work that has been contributed by midwives, general practitioners, medical officers of health, resident medical staff of all grades, anaesthetists, consultants and Ministry of Health personnel—has assuredly been fully justified. Of relatively minor importance are the possible faults and lapses shown to have occurred in the past, for they are undoubtedly eclipsed completely by the good that must inevitably follow in the future. Lessons

there are to be learnt and attention has been directed to some of the more important. Some will influence the individual—the practitioner of obstetrics—and others concern the administration of the midwifery service. The two are indeed complementary one to the other, but the former is by far the more important, for no amount of good administration can remedy poor obstetrics.

To all who practise obstetrics comes a dual message. In the first place, the truly remarkable fall in the maternal and perinatal mortality rates over the last 2 decades must be regarded as a genuine reward for care of the woman in the prenatal period and of increased skill in her supervision during labour and subsequent delivery. Admittedly the satisfactory results have been aided by what may be termed The General Advance in Medical Knowledge. In the second place, the report stresses the utmost importance and enormous value of prenatal care. No evidence was forthcoming that any part of this work is redundant, nor was there found a suggestion that we might possibly relax our efforts in one single direction. On the contrary, the Report has brought before us weak spots which deserve our attention and certainly merit repetition. To the individual doctor, be he in general or specialist practice, comes the warning that in antenatal care it is possible to pay insufficient attention to the insidious onset of anaemia or of the early symptoms and signs of pre-eclampsia, and that there is no place whatever in these conditions for a policy of *laissez faire*. Their detection indicates immediate and effective action. The same warning is justified in all pregnant women suffering from valvular disease of the heart. In all antenatal work, whereas regular observations made at accepted intervals are of value, non-attendance of the patient merits immediate investigation. In the conduct of the labour, and especially in the management of the third stage, there is no justification for delay in sending for additional help. There would appear to have been an occasional lack of appreciation of the value of blood transfusion in this respect and also of the services of the emergency obstetric team.

To all upon whom rests the responsibility for making a decision as to the necessity and performance of Caesarean section, this Report suggests that it is possible to give insufficient thought to the question whether the general state of the patient is as satisfactory as is possible before the operation is commenced. Also, during and following the operation, a warning is apparent that there are times when severe blood loss seems to have been looked on as inevitable and has not been followed by immediate replacement. Lastly, the Report suggests that our colleagues, the anaesthetists, might ask themselves whether they could not direct some of their skill and research towards increasing the safety and ease of administration of anaesthetics to women in labour.

In regard to administration, the first and most certain requirement surely is the absolute necessity for an organized service. In Britain this has been suggested by our Royal College twice during the last 10 years, and each yearly report has suggested a simple organization for the supervision and structure of such a service. It seems unlikely, however, that we shall put our house in order in any manner more rapid than the usual process of evolution. The availability and distribution of beds for obstetrics is also a matter for administration, and in this respect emphasis must be made on the fact that the number of beds available for antenatal cases must depend on the population to be served and not on the size of the obstetric unit existent in any locality. The organization and distribution of the Emergency Obstetric Units is now a matter of proved importance, whether their transport be by land, water or air.

How interesting it is, after the above paragraphs have been written, to read the recent article by Jewitt[1] to which reference has already been made, in which he states that the findings of his investigation, which differ very little from those in the Report we are considering, reveal 'five imperative demands upon the profession:

1. Doctors and public alike must be educated to better antenatal care.
2. Transfusions must be used early and copiously.
3. Caesarean section should be avoided at all reasonable cost.
4. A fanaticism of asepsis should be resumed to combat the rising proportion of septic deaths. (So far this is absent in Britain.)
5. Obstetricians must demand better anaesthesia.'

In the summary of the Report occur the following words: 'Reviewing the clinical and statistical information in the study of this series of maternal deaths it would seem justifiable to draw attention to the following points which should influence obstetric practice. (Only the headings will be given, in the order in which they are presented.):

I. Nearly one-quarter of potential avoidability in maternal mortality lies in the patient's refusal or neglect to follow medical advice or to seek advice.
II. The first step in the care of every pregnant woman should be careful consideration of the arrangements for confinement.
III. The next step should be to plan the pattern of antenatal supervision at the onset.
IV. In antenatal care the first consideration should be the assessment of the woman's general health.
V. Refers again to unsuitable arrangements for confinement and lack of appreciation of the use of the emergency obstetric team.'

While all these are correct, it is possible to summarize in a different manner. Reference has already been made to the lowly position now taken by puerperal sepsis as a fatal illness, and all who are familiar with the thousand and more reports were equally impressed with the rarity of what may be termed disasters in labour such, for instance, as the case of failed forceps. In consequence, but little help would be obtained in an effort to discover causes for maternal death which possessed an avoidable factor in these two groups. Thirty years or more ago this would certainly not have been found to be so, and perhaps our greatest lesson is to be learned in a brief consideration of how this great change has been brought about. Puerperal sepsis is no longer the terror of midwifery, partly because the haemolytic streptococcus has decreased in virulence and partly owing to the discovery of antibiotics. The diminished risk of death from disasters occurring in late labour presents a more difficult problem. No doubt the advent of antenatal observation, the steady increase of hospital confinement, where skilled help and nursing is more readily available, the use of X-rays, and a fuller education of medical students, have all contributed to greatly better results. Viewed in another direction, throughout this series of cases, no matter whether pre-eclampsia, haemorrhage, heart disease, or whatever other

condition be taken to be the cause of death, the importance of the general health of the woman before and during pregnancy and labour stands out immediately as a factor of the utmost importance. Whereas this has been appreciated in some degree by a few, it has seldom received more than lip-service by doctors and midwives generally, whether they work independently or in clinics. Antenatal care in its early days, and to a great extent up to the present time, has interested itself for the most part in what may be termed the mechanistic aspect of obstetrics. By this is meant the detection of a contraction of the pelvis (and nowadays owing to the virtual disappearance of rickets the deformity is usually slight), the discovery of disproportion, or the finding and attempted correction of an unfavourable presentation or position of the foetus. All these efforts are praiseworthy and must continue, but their value is limited. In their detection it is as though the enthusiasm for them has been such as to lessen the observance of something else that is surely far more important, namely the patient herself and the state of her health.

It would almost be fair to say that, given a woman of normal stature and in a state of good physical health, the potential dangers of what has been called the mechanistic aspect of prenatal care almost vanishes into thin air. Such a patient will not have a contracted pelvis and, provided the foetus is of normal size, she will deliver herself safely and unaided no matter what is the foetal presentation or position. That this is true has been shown recently by the work of Dugald Baird[3,4] and others. To ensure normal build and stature, and with it good physical health, the care of an individual must start in infancy or even before birth and must be continuous throughout childhood and adolescence. This indeed has been done in Britain and in many other parts of the world and has virtually abolished all but minor degrees of pelvic contraction. But it is not appreciated by what we may term 'the antenatalists'. To them the importance of the non-engaged foetal head is such as to mask a state of debility, gross dietetic errors, anaemia, early signs of heart failure, or indeed any deviation from normal good health.

The absolute necessity for a ceaseless, faultless and painstaking vigilance for the detection of the earliest symptoms and signs of pre-eclampsia has already been noted. Failure when it occurred seems to have been due to a lack of appreciation that the earliest symptoms and signs of pre-eclampsia indicates the necessity for action in arranging at once for the patient's admission to hospital, where continual observation and assessment presents no difficulties. In other words, the emphasis is on continuous and meticulous care in the preservation of good health.

This aspect of the case is just as important in the matter of loss of blood. A moderate haemorrhage in a woman in good health may not be dangerous, whereas if she is not in good health, and especially if she is anaemic, the same haemorrhage may be fatal. The risk of anaemia developing in pregnancy is very real. Far too often no serious attempt is made at any time to assess the blood state of the patient. Even if this is done, there seems to be little appreciation of the vital importance of treatment so active that it will ensure that the woman reaches the end of her pregnancy with a normal blood picture. In the series of cases of haemorrhage there were instances also of failure to replace blood lost, or to be too slow, for varying reasons, in so doing. All these were especially noticeable in cases of Caesarean section. Once again the importance of the general physical state takes first place.

Valvular disease of the heart ranks high as a cause of maternal death and it is suggested that the condition may be presented as yet another that receives insufficient attention simply because it represents a deviation from what may be termed the general, rather than the obstetric, state of the patient. This theme could be continued almost indefinitely, but the picture would be the same. The examples that have been given are only examples of single items. The production and maintenance of that often ridiculed state of 'positive health' has only of late years received much attention. It may well be that exhaustive and exhausting research into the almost unexplored regions of dietetics will in the course of time provide a major, if not final, solution to the cause and abolition of many illnesses, and therefore dangers, peculiar to pregnancy and childbearing.

It is opportune at this point to direct attention to another and extremely important benefit that inevitably must accompany meticulously careful prenatal work. Whereas it will be somewhat grudgingly accepted that such measures may be expected to be followed by a small drop in an already exceedingly low rate of maternal mortality, it should never be forgotten that for every registered maternal death there are inevitably a far greater number of women who suffer but survive, and about whom nothing exact is known. They could be classed under the heading of maternal morbidity. Any reduction in maternal mortality, therefore, will result in a correspondingly larger reduction in maternal morbidity. Last, and by no means least, maternal mortality and maternal morbidity are equally certainly related to the perinatal mortality of the foetus. It may well be that every effort to reduce the maternal mortality rate will be productive of the heaviest dividend of all, namely, a truly significant diminution in stillbirth and neonatal death rates.

ENVOI

The closing note of this review, therefore, is to give emphasis to what (in my opinion) is the most important lesson to be learnt from the Report. The suggestion is made that as the result of many pointers, of which this recent Report on maternal deaths is one, consideration should be given to the advisability of an altered outlook towards prenatal care. While in no way diminishing the observance of and search for what may be termed purely obstetrical abnormalities, there should be a much wider appreciation of the relatively greater importance of the general health of the woman. Put in other, if rather unkind, words, we should regard our patients as patients, and not as travelling incubators whom we inspect at regular intervals for the detection, and if possible rectification, of some fault in function of relatively minor importance. Briefly stated, let the obstetricians tend to return toward the ranks of the general physicians and as such to practise Medicine with a capital 'M' rather than to wander, if not stray, into ever increasing specialization.

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SACRO-ILIAC SYNOSTOSIS AND PELVIC DEFORMITY

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Recently, while working through a collection of bones the records of whose origin had been lost, I came upon an adult female pelvis showing unilateral sacro-iliac synostosis associated with a typical oblique contraction ('Naegele' pelvis). The obliquely contracted pelvis has usually been regarded as very uncommon (Baird, 1950). However, Greenhill (1955) says: 'This type of pelvis is more common than is usually believed. Thoms in routine roentgen studies of obstetric patients found 3 cases in 2 years'. On this account it has seemed to me that a brief account and discussion of this specimen would be of value.

The pelvis is that of a mature but at most middle-aged woman; the character of the pubic symphyseal surface, judged by the criteria laid down by Todd (1921) for American White and Negro-white hybrid women, suggests an age above 30 but probably less than 50 years. The lumbo-sacral and left sacro-iliac joints show very slight accentuation of their margins, but no osteophytic outgrowths. The front of the bodies of the 1st and 2nd sacral vertebrae is, however, very much roughened over the whole area for attachment of the anterior longitudinal ligament. The acetabula are symmetrical and free from pathological changes.

Fig. 1 shows the typical 'Naegele' distortion of the outline of the pelvic brim. The sagittal plane of the sacrum, if prolonged forwards, passes to the right of the right pubic tubercle, so that the symphysis lies fully an inch to the left of this plane. The iliac crests are, however, approximately symmetrical. The principal dimensions of the pelvis are as follows: Intercristal diameter 245 mm., anterior inter-spinous diameter 215 mm., sagittal diameter of brim from promontory 92 mm., promontory-symphyseal conjugate 98 mm., transverse diameter of brim 119 mm., right oblique diameter 125 mm., left oblique diameter 108 mm.

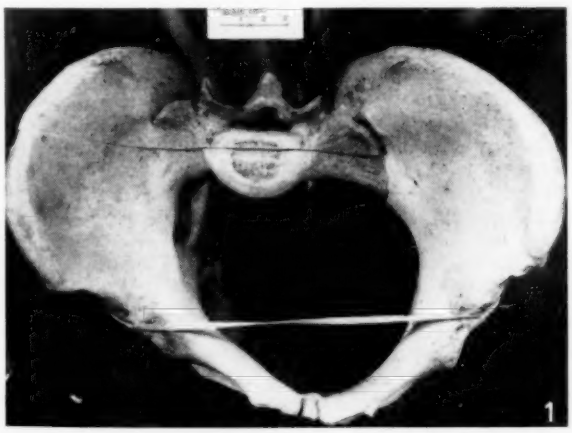

Fig. 1. The pelvis described in the text, viewed perpendicular to the plane of the pelvic inlet. The left iliac spine, which is in deep shadow, has been outlined in white.

The right sacro-iliac joint is solidly synostosed. On the iliac face its line is marked by a slight and irregular bony ridge; on the pelvic face it cannot be traced at all, a completely smooth surface flowing from the ilium on to the lateral mass of the sacrum. There is no trace of a pre-auricular sulcus on this side, whereas there is a slight and narrow one on the left. Posteriorly the ossification process seems to have involved the interosseous ligament to some extent. Although the line of the joint cannot be completely traced, it is clear that the lateral mass of the sacrum does not extend so far laterally on the right side as on the left. The great sciatic notch is slightly narrower on the synostosed than on the normal side; the latter admits 3 fingers with ease, the former with slight difficulty. On the normal side there is a conspicuous roughened area at the junction of the iliac and pubic parts of the pectineal line; on the fused side this is only faintly developed.

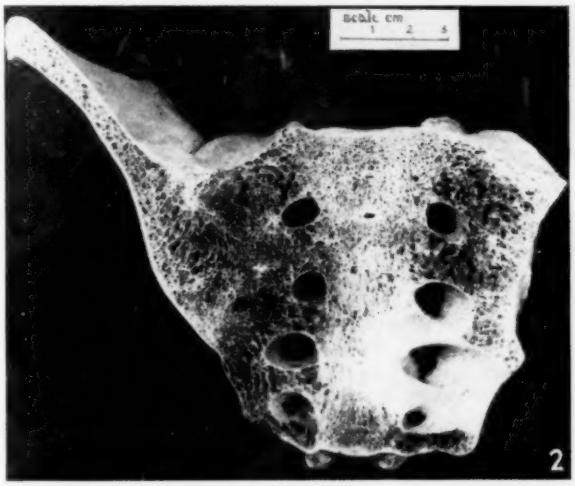

Fig. 2. Section through the sacrum and right ilium in the long axis of the sacro-iliac joint.

A saw-cut has been made through the specimen approximately in the long axis of the sacro-iliac joint. As Fig. 2 shows, there is no trace of a joint cavity in this section. The slight ridge which marks the position of the joint on the iliac face is formed by a thickening of the outer compact stratum; at the inferior limit of the joint there is no such thickening. The internal structure is a meshwork of cancellous bone. Superiorly and inferiorly the presumed position of the joint is marked by a zone of relatively fine mesh, but this thins out and disappears towards the centre, where the coarser meshwork extends without a break from the ilium into the sacrum.

The section through the auricular surface on the normal

side of the sacrum shows that the skin of compact bone underlying the articular cartilage is very thin. It is backed by a narrow zone of fine-meshed cancellous bone which merges into the coarser meshwork of the interior.

TWO PROBLEMS

The problems raised by this specimen are, first, the relationship between sacro-iliac fusion and pelvic asymmetry, and second, the cause of fusion in this instance.

Heyns (1955) points out that additions to the perimeter of the pelvic brim take place at the acetabular, the symphyseal, and the sacro-iliac joint epiphyseal cartilages. The epiphyseal plates for the auricular surfaces of the sacrum are responsible for the lateral growth of the sacral alae; it is not clear whether the amount of bone growth on the innominate surfaces is comparable. Despite minor complexities, it is clear that increase in the width of the posterior sacral segment depends very largely upon growth at the sacro-iliac joint, chiefly it would seem on the sacral side. Heyns further suggests that growth along the ventral border of the iliac auricular surface is associated with a migration dorsally of the sacro-iliac joint. Differential growth along this border may be responsible for the sex difference in inclination of the sacrum, and indirectly for that in width of the great sciatic notch.

Payton (1935) suggested that the 'Naegele' deformity might result from failure of the sacro-iliac joint on one side to migrate dorsally. This would manifest itself by an unusual distance between the joint and the crest of the ilium, as though the ilium had grown past the sacrum instead of carrying it along. In the present specimen, however, the two ilia are approximately symmetrical in relation to the dorsum of the sacrum. In spite of the slight difference in the sciatic notches, it seems very unlikely that the deformity in this case can be attributed primarily to failure of the sacro-iliac joint to migrate dorsally.

The most probable cause of the deformity in this case is therefore a failure of growth on one side of the sacrum. This explanation of the 'Naegele' and its relative the 'Robert' pelvis is very generally accepted, but on the reason for the sacral defect and its relation to sacro-iliac fusion there is no such agreement. One school of thought, represented by Baird (1950), considers that the 'Naegele' deformity usually results from disease of the sacro-iliac joint in infancy with ankylosis; the alternative view, expressed by Brailsford (1953), attributes it to failure of development of the ala of the sacrum on the one side and subsequent fusion of the ilium to the defective sacrum. Greenhill (1955) admits both possibilities, but maintains that congenital defects of the wings of the sacrum do not produce so gross a deformity as disease of the sacro-iliac joint.

It is understandable that disease of the sacro-iliac joint in childhood, with more or less complete destruction of the growth cartilage, should be followed by ankylosis, and that subsequent growth at the other cartilage zones would produce increasing deformity. Why a primary defect in the sacral ala should tend to sacro-iliac fusion is less obvious.

My former colleague in the University of Edinburgh, Dr. Ethne Little, had the opportunity some years ago of studying a young adult woman displaying a typical 'Robert' pelvis. In this case there was radiologically no evidence of sacro-iliac fusion, although the classic descriptions of the Robert pelvis insist on its presence. There seemed to be no reason for dissociating this case from other examples of the Robert pelvis. The conclusion to which it pointed appeared to be that the primary defect is in the growth of the sacrum. From this point of view it might be argued that the Robert pelvis is an extreme degree of the 'anthropoid' pelvic form. Whether these inferences can be extended to cover the Naegele pelvis is another matter.

Complete unilateral fusion without pelvic asymmetry has been described by Shore (1930) in a Zulu male aged 56 years. On section, the position of the joint was found to be marked by a band of compact bone several millimetres broad and remarkably even in width. The cancellous bone on either side of this band was very regular in its organization, with no evidence either of excess bone formation or of bone absorption.

Shore argues that 'uniformity in the extent of the union, uniformity in the compact bone which replaces the sacro-iliac joint, and uniformity in the texture of the cancellous bone which abuts on the union are jointly and severally inconceivable as the result of synostosis of arthritic origin'. He therefore regarded his specimen as one of 'developmental' or non-pathological synostosis. From the symmetry of the pelvis he concluded that fusion had taken place after growth was completed, inferring that fusion in early life would have resulted in asymmetry. Further, he tentatively suggested that the synostosis might be likened to the fusion of an epiphysis or of the vertebral components of the sacrum. This suggestion clearly cannot be pressed too far, since a synovial joint is concerned and not a simple cartilaginous layer.

Sacro-iliac fusion, however it comes about, involves an obliteration of the joint cavity. It is indeed conceivable that the process of chondrification might extend across the primitive joint plate so that no synovial cavity developed. Secondary fusion between the two articular cartilages is perhaps easier to envisage, even in the absence of distinct pathological changes. Whillis (1940) indeed claimed that adhesion between articular cartilages is frequent if not normal in late foetal life, but his evidence has not been substantiated by other investigators.

Schunke (1938) states that cavitation in the sacro-iliac joint begins during the 10th week of intra-uterine life, but does not reach its full extent until late in the 7th month, although passive motion commences after the 6th month. Fibrous strands connecting the articular cartilages are present in foetal and early post-natal life and may be found in the adult. Brooke (1924) describes fibrous adhesions between the articular surfaces in the adult, giving the joint the semblance of a fibro-cartilaginous amphiarthrosis, but he insists that this is a pathological condition associated with advancing age. Schunke finds that the synovial cavity is normally preserved throughout life, even when extra-auricular synostosis has occurred. This type of union, which is common in the 5th decade especially in males, is due almost entirely to ossification in the anterior sacro-iliac ligament above the pelvic brim (the proximal sacro-iliac ligament of Fick). The condition, of which I have seen a number of examples, is quite different from true sacro-iliac fusion.

The specimen described by Shore presents the impression

that the joint space has become completely filled by compact bone. In mine there is no such band of compact bone, merely a discontinuous zone of fine-meshed cancellous bone. This might simply mean that the fusion is of longer standing in my specimen, but there is no way of proving this.

Although there is no gross evidence of inflammatory reaction in my specimen, I do not think that a pathological background can be excluded. Indeed a pathological destruction of the joint and the adjacent growth cartilages during childhood, followed by bony repair, is in many respects the most intelligible explanation.

From the foregoing it will be evident that no final answer to many of the questions raised by this study can as yet be given. It is clearly necessary for cases showing either defective sacral development or juvenile sacro-iliac arthritis to be followed through life and if possible to autopsy. The difficulty is that in many cases the anomaly, if not discovered accidentally in the course of some other investigation, may betray itself only as a well-established deformity or may pass unrecognized throughout life.

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COLD HAEMAGGLUTINATION IN PRIMARY ATYPICAL PNEUMONIA

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Cold haemagglutination has been the subject of investigation by many workers, and has been shown to occur in healthy human beings as well as in many animals. It has also been demonstrated in association with many diseases, usually to a much higher titre than in normal, healthy people. Li-Chen-Pien[1] described it in a patient suffering from hypertrophic cirrhosis of the liver. It has been found in association with leukaemia by Alexander and Thompson,[2] Boxwell and Bigger,[3] and Manheims and Brunner.[4] Dameshek[5] mentioned its association with infectious mononucleosis, and Stats and Wasserman[6] and Muschel[7] described its occurrence in pregnancy. Increased titres of cold haemagglutinins, however, have been found consistently only in trypanosomiasis (York[8]) and in primary atypical pneumonia.

Clough and Richter,[9] in 1918, were the first to describe a raised cold haemagglutinin titre, in association with a condition which would now be diagnosed as primary atypical pneumonia. They did not associate this serological abnormality with the respiratory condition, and thought it to be coincidental. Since 1943, however, numerous references have been made in the literature to this association between raised cold-haemagglutinin titres and primary atypical pneumonia. Turner *et al.*[10] investigated 83 cases of primary atypical pneumonia. Of these, 44 (53%) had titres of at least 32, and 23 patients had titres of 128 or higher. In their control series, consisting of 132 patients suffering from various other diseases, in only 5 (3·7%) did the titre exceed 16. Horstmann and Tatlock[11] investigated 43 cases and found an incidence of 67·5% of significantly raised cold-haemagglutinin titres. Peterson *et al.*[12] mention that the great majority of the patients with primary atypical pneumonia which they had encountered showed cold haemagglutinins in dilutions of serum ranging from 1/10 to 1/10,000. Young,[13] in studying a large series of cases, reported an incidence of over 51% in which the titre was 128 or higher, whilst his normal controls and other respiratory diseases showed an incidence of only 0·7%. Finland and Barnes[14] investigated 676 patients suffering from a wide variety of acute respiratory infections. They came to the following conclusions: (1) The great majority of uncomplicated cases of primary atypical pneumonia develop significantly raised cold-haemagglutinin titres; (2) the demonstration of high-titre cold haemagglutinins is almost always associated with primary atypical pneumonia, although the finding of this disease was sometimes obscured by complicating conditions; (3) patients with acute respiratory infections other than primary atypical pneumonia fail to develop high-titre cold haemagglutinins; (4) the demonstration of high-titre cold haemagglutinins is of considerable value in the diagnosis of primary atypical pneumonia.

The spate of articles describing the occurrence of high-titre cold haemagglutinins in primary atypical pneumonia appearing in the medical press in 1943 and at sporadic intervals afterwards originated mainly from the USA. In virtually every article the significant incidence of high-titre cold haemagglutination was stressed. It has, in fact, become customary for cold-agglutinin investigations to be carried out as an aid to diagnosis in cases of atypical respiratory infections.

The demonstration of high-titre cold haemagglutinins in primary atypical pneumonia, or any respiratory infection for that matter, is a rare occurrence in the experience of our laboratories during the past 11 years, and it is probable that physicians in this country have been struck by the monotonous regularity with which such investigations have given negative results. The investigations described in this paper were carried out in order to confirm these findings.

The differentiation between 'normal' cold haemagglutinins present in many normal, healthy people, and 'abnormal' cold haemagglutinins associated with disease, depends to a large extent on the concentration in which the cold haemagglutinins are present in the serum. The exact dividing line between

'normal' and 'abnormal' cold haemagglutinins is debatable, although a titre of 16 is the most generally accepted upper limit of the normal range. This figure is questionable; many workers consider the upper limit of normal to be 128, and titres as high as 500 have been described in apparently normal, healthy people (Kyuhei Nakadate[15]). The confusion is increased by variations in technique. It is probable that before definite pathological significance can be attached to cold haemagglutinins the titre would have to be well in excess of 16, but for purposes of this investigation the latter figure was accepted as the upper limit of the normal range.

Cold-haemagglutinin technique:

A clotted specimen of the patient's blood is incubated at 37°C for 2 hours to allow elution of cold haemagglutinins to take place. The serum is separated immediately after the specimen has been removed from the incubator. Twofold serial dilutions of the serum in saline are prepared, and a drop of each dilution is added to each of two tubes. One drop of a washed 2% suspension of the patient's cells in isotonic saline is added to each tube in one row of serum dilutions, and one drop of freshly prepared, washed 2% suspension of group O cells in isotonic saline is added to each tube in the other row. The cells and diluted serum are mixed, and then refrigerated at 4°C for 12-18 hours. The results are read macroscopically, and scored from ++++(solid agglutinate) to +(visible granularity). The highest titre at which visible granularity can be detected is taken as the end-point of the titration.

The investigation consisted of a 160 patients tested for cold haemagglutinins (Table I). Of these patients 27 had been diagnosed clinically as virus or primary atypical pneumonia, 33 as pulmonary tuberculosis, 12 as some other pulmonary infection (lobar pneumonia or bronchopneumonia), and 88 as suffering from various non-respiratory conditions. It is apparent from an analysis of these figures that the incidence of high-titre cold haemagglutinins in primary atypical pneumonia (7.6%) is insignificant when compared with figures quoted by American authors, in which the incidence ranged from 51% to 67.5%. The incidence compares more

TABLE I. 160 PATIENTS TESTED FOR COLD HAEMAGGLUTININS

	Primary atypical pneumonia	*Pulmonary tuberculosis*	*Other respiratory conditions*	*Various non-respiratory diseases*
No. of cases investigated ..	27	33	12	88
No. in which cold haemagglutinin titre exceeded 16	2	1	0	2
Incidence	7·6%	3%	0%	2·3%

favourably with that of their control series, consisting of various respiratory infections other than primary atypical pneumonia. The records of cold-haemagglutinin investigations carried out at the Blood-Group Research Laboratories from 1951 to 1954 were also analysed (Table II). These results are in close agreement with those of Table I. The final analysis is therefore based on 590 investigations comprising 79 cases of primary atypical pneumonia, 33 cases of pulmonary tuberculosis, 33 cases of other types of respiratory infections, and 445 patients suffering from various non-respiratory diseases (Table III). The most important point which emerges from an analysis of these findings is that the incidence of

TABLE II. ROUTINE INVESTIGATIONS CARRIED OUT AT THE BLOOD-GROUP RESEARCH LABORATORIES 1951–54

	Primary atypical pneumonia	*Pulmonary tuberculosis*	*Other respiratory conditions*	*Various non-respiratory diseases*
No. of cases investigated ..	52	0	21	357
No. in which cold haemagglutinin titre exceeded 16	0	—	2	1
Incidence	0%	—	8%	0·28%

high-titre cold haemagglutinins in primary atypical pneumonia (2.6%) is low, being similar to the incidence in other types of respiratory infections (4.5%), in contrast to the highly significant incidence described by American workers. It would appear therefore, that the presence or absence of high-

TABLE III. FINAL ANALYSIS—588 INVESTIGATIONS

	Primary atypical pneumonia	*Pulmonary tuberculosis*	*Other respiratory conditions*	*Various non-respiratory diseases*
No. of cases investigated ..	79	33	33	445
No. in which cold haemagglutinin titre exceeded 16	2	1	2	3
Incidence	2·6%	3%	6%	0·67%

titre cold haemagglutinins is of no value in diagnosing the nature of a respiratory infection. These remarks are certainly applicable to the Witwatersrand area, from which most of the specimens used in the investigation originated, and presumably also to the rest of Southern Africa.

It should be pointed out that the specimens used in this investigation were submitted to our laboratory for routine investigation, and originated from many different sources. It is certain that the diagnostic criteria used in the diagnosis of primary atypical pneumonia, being the enigma that it is, were not uniform, and that in some instances the condition did not conform to the clinical entity described by American workers. Some of the patients probably suffered from Q fever or one of the lesser known diseases which may simulate virus pneumonia. As the analysis, however, included all specimens investigated by our laboratory since 1949, one cannot but come to the conclusion that either the incidence of virus pneumonia is not significant in this country or, if it is, then the disease process must in some way differ from that occurring in the United States.

Macaulay[16] considers cold haemagglutination to be a non-specific response to lung-tissue destruction, occurring in all types of lung disease, although most commonly in primary atypical pneumonia. In the light of the above findings it seems much more likely that the development of high-titre cold haemagglutinins can be correlated with the aetiological agent, presumably a virus[17] different strains of which would

be found in various geographical regions, and only some of which are capable of stimulating the formation of cold haemagglutinins.

SUMMARY

An analysis of cold-haemagglutinin investigations carried out in 79 patients suffering from primary atypical pneumonia, 66 patients suffering from other respiratory conditions, and 445 patients suffering from various non-respiratory diseases, show that there is no significant difference in the incidence of high-titre cold-haemagglutinins in primary atypical pneumonia and other respiratory infections and that, in South Africa, the demonstration of the presence or absence of high-titre cold haemagglutination is of no value in diagnosing the nature of a respiratory infection.

The mechanism of cold haemagglutinin formation is briefly discussed.

This investigation was made possible by the kind permission of the Director of the South African Institute for Medical Research. I am also grateful to Dr. A. Zoutendyk for his helpful criticism, and for allowing me access to the records of his department.

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SOME PROBLEMS OF THE EXTRACORPOREAL CIRCULATION*

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The problem of the 'dry heart' has been with us for many years and any progress in this subject is bound to bring with it an increase in our knowledge of the circulation. At St. Thomas's Hospital in London for the past 5 years my colleagues Drs. Kent Harrison and MacMillan and I have had a modest research programme devoted to learning something of the difficulties imposed by the interruption of the circulation.

Up to 2 years ago our energy was devoted to developing and learning something of the limitations of the technique of surface cooling in hypothermia. The reduction in cellular metabolism appeared to be an ideal method of achieving a temporary interruption of the circulation. It is true to say that hypothermia has not lived up to our original expectations. The main problem is to explain the reason for the increased irritability of the myocardium as the temperature falls. There are 4 favourite theories concerning factors which may account for this, but in none is the evidence conclusive.

1. Anoxia—low oxygenation of the coronary blood.
2. Electrolyte Changes, particularly rise in ionized calcium.
3. Alterations in the pH of the Blood.
4. Excess of Catechol Amines i.e. adrenaline or noradrenaline.

Today, hypothermia is used clinically down to 30°C (normal, 37°C) and this allows the surgeon approximately 10 minutes in which to carry out some intracardiac procedure. Meanwhile our attention has been turned to the other possible line of approach to this problem, namely the extracorporeal circulation.

I feel it would be very wrong to create the impression that we have been or are contributing anything original in this field. Our aim has to be to learn the difficulties involved, and as there are so many little problems it seems clear that progress will be made only if each group of workers fully discusses its failures.

There are two big subdivisions in the type of mechanical pump that can be used. In the first, the principal aim is to supply only a limited quantity of oxygenated blood to the brain and vital organs, and no attempt is made to ensure an adequate flow. These machines are called 'low-flow oxygenators' and the Lillehei machine is the best example. The principle is based on the finding of Andreasen and Watson (1953) that at normal body temperature the dog requires only the very low flow of about 50-60 ml. per minute to maintain life during a period of occlusion of the heart from the circulation. Dr. Lillehei with his bubble oxygenator has applied these findings to man and has now performed many successful operations with this technique. Our experience with this type of oxygenator was very satisfactory but it was difficult to obtain more than 200-400 ml. of oxygenated blood per minute. Physiologically it would not seem correct to supply a very reduced flow of blood to the vital organs, but rather to try to perfuse all the body with the same flow rates as it would normally receive. Here the problem immediately grows, and machines of the 'high-flow' type are more complex.

I have had the honour of working in Dr. John Gibbon's laboratory in Philadelphia, USA. For nearly 15 years now he has pioneered the development of a machine capable

* A paper presented at the South African Medical Congress, Durban, September 1957.

of handling the whole circulation, and the recent reports of the application of this machine for human cases at the Mayo Clinic have more than justified the enormous effort that has been made. The Gibbon machine is extremely complex but essentially it consists of a metal screen oxygenator and rotatory squeeze pumps. In England we are using the Melrose pump, which incorporates compression pumps and a rotating disc oxygenator. Whatever the machine, the problems are still the same. First and foremost is the training of a team that is familiar with all the problems involved. In this type of work the days of the one-man band are over, and progress will only be made by a conjoint effort.

There are a certain number of factors involved that, in the light of our own experience, deserve special mention.

Donor blood. In human cases this is no problem because there are ample supplies of fresh blood available. The oxygenator and pump must be primed with freshly taken heparinized blood. In dogs the supply position is far more difficult and a mixed supply of mongrel blood appears to be better than that of a single pedigree donor such as a greyhound. The blood must be freshly taken and should not be stored, for the use of stored blood leads to continued bleeding at the end of the experiment.

Blood volume. Minute care must be taken to assess any alterations in blood volume. All quantities infused or lost through haemorrhage must be calculated and recorded. In nearly every case there is a distinct tendency to overload the circulation with fluid.

Blood flow. In animal work the blood flow per minute is related to body weight for simplicity, but in man it should be represented as the number of millilitres per minute per square metre of body surface.

In dogs, a good perfusion is one in which the flow is great than 70-80 ml./min./kg., i.e. 700 ml. per minute for a 10 kg. dog. In man the ideal flow rate is 2·2-2·5 litres per minute per square metre of body surface (Kirklin *et al*, 1957). Flows lower than this tend to cause inadequate perfusion of the tissues and a severe metabolic acidosis develops during and after the perfusion.

Venous drainage is best performed by gravity drainage rather than by mechanical sucking and the optimum height can be calculated before the pumping commences. Too great a venous suction will only result in collapse of the vessel walls. The venous catheters are placed one in the superior vena cava through the azygos opening and one in the inferior vena cava through the right atrium. The aterial perfusion in dogs is done through the carotid artery, and in man through a branch of the subclavian.

Blood coagulation. The animal is heparinized on the basis of 2 mg. of heparin per kg. of body weight. Each pint of blood is taken into a solution containing 25 mg. of heparin and 50 ml. of 5% dextrose solution. At the cessation of the artificial circulation the animal is given 2 mg./kg. of protamine sulphate to aid clotting. After a few minutes an intravenous infusion containing 100 ml. of saline with a further 2 mg./kg. of protamine is commenced at a very slow rate and the total quantity is given over the next 3 hours.

Air emboli. Particular care is taken to siliconize the whole machine before commencing the experiment and then to circulate the blood well before use to remove any small air bubbles that might be adhering to the tubing walls. A filter of some type is necessary to ensure that no fine clots are injected.

Significance of arterial and venous pressure. Both are important measurements which reflect on the progress of the artificial circulation. A rise in venous pressure during the circulation suggests that there is a partial obstruction to the venous catheters and this must be remedied immediately. After the pumping, a rise in venous pressure indicates either a failing myocardium or overloading of the circulation with fluid.

Anaesthesia. One of the principal features of all cases involving extracorporeal circulation is the prolonged time taken both in the dog and in man to recover consciousness. It is important, therefore, to use only the minimal anaesthetic requirements and, in particular, those that can be eliminated rapidly at the end of the circulation. The possible causes of failure, and thus death of the experimental animal, are legion in this type of research. Yet after training and removal of obvious errors of technique there still remains a disturbingly high mortality. As yet no one major factor has been elucidated. In the first 12 hours after the conclusion of the experiment the animal requires constant attention to replace haemorrhage, sustain the systolic pressure, and prevent pulmonary collapse and many other hazards that lie in wait.

To end on a personal note, I do not think I know of any other type of experimental work which is so completely and continuously demanding of our attention and yet is so often attended by a frustrating result. This can only mean that we have a lot to learn.

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OPEN HEART SURGERY*

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Open Heart Surgery is being performed in many centres in the United States today. The thoracic surgeon who intends to study these modern methods must of necessity limit his visits to the best known of the Clinics.

Accordingly, for reasons which I shall discuss later, I divided the precious time at my disposal between Philadelphia, where Charles Bailey was in charge of the surgery, Minneapolis, under the aegis of Lillehei and Varco, and the Mayo Clinic at Rochester where Kirklin was the presiding genius.

My main interest naturally was in watching the surgical techniques and the special anaesthetics they required, but I had hoped to glean some information on the diagnostic methods. Unfortunately I was not able to see many of the pre-operative investiga-

*A lecture given to the Southern Chapter (S. Africa) of the American College Chest Physicians.

tions, nor the post-operative 'follow-up', as I could not spend longer than 14 days at each of the selected centres.

We have witnessed wonderful progress in heart surgery during the last 10 years. Many patients, for years under medical treatment alone, are now being helped and even cured by surgical techniques.

The exploration of the interior of the heart had always been the deterrent to further advances. Conditions such as patent ductus arteriosus, coarctation of the aorta, constrictive pericarditis, mitral stenosis, and some of the congenital abnormalities of the aortic arch had all proved amenable to surgery, but opening and inspection of the heart was not necessary in these cases. Disease processes inside the heart, such as mitral stenosis, aortic stenosis, and pulmonary stenosis, valvular and infundibular, were treated by blind measures, i.e. instruments were inserted into the heart and the surgery was performed while the heart was in action.

The latest and most exciting advance was the so-called 'Open Heart' surgery, which allows the surgeon to operate under direct vision. This necessitates alteration of the heart function in some way, so that the normal circulation and oxygenation of the blood can be maintained during the operation.

BAILEY'S CLINIC AT PHILADELPHIA

The name of Dr. Charles Bailey is associated with every branch and facet of cardiac surgery, and in Philadelphia I learned that not only is he an extremely able surgeon but a man whose nimble and original mind has devised many instruments used for the different aspects of heart operations. My first introduction to heart surgery was thus the profitable period I spent with Bailey and his assistants at the Hahneman Hospital in Philadelphia, and I shall tell you of a few of my experiences there. I was surprised at Bailey's operating theatres, as I had always believed that most of the American specialist hospitals were wonderfully equipped, more or less as depicted in the cinema operating theatre scenes. This was certainly not at all the case here, where Bailey performed wonderful work with the primitive and inadequate facilities offered by this old hospital.

As most of you are aware, the operation for mitral valvotomy is carried out through the left side of the chest. Bailey, however, operates through the right side, his reasons being that not only do his patients suffer from mitral stenosis, but 30% of all his cases with mitral stenosis also have some degree of tricuspid stenosis. In order to reach both the mitral and tricuspid valves, he considers that the right side offers the best approach. Naturally I quote Bailey's own statement that 30% of his cases have tricuspid stenosis as well as mitral stenosis, and he adds—to strengthen his case—that there are cases in which there is co-existing aortic stenosis, so that the patients require what is referred to as a 'three-valve-job'.

I should like to describe Bailey's right-sided approach for mitral valvotomy. A plane is found between the right and left atrial walls, and when the tissue has been incised for a short distance between these two walls, the left atrial cavity is entered from behind the heart. He maintains that this access gives him a good opportunity to feel the entire mitral valve. In my opinion this is not only a more dangerous but also a more difficult procedure, although it does not appear to be so in his hands. Having carried out the mitral valvotomy, a variety of valvotomes designed either by his assistant or himself are used, and he then explores the right atrial cavity. If necessary, the aortic valvotomy is then performed by means of a trans-aortic approach. His operative results are good, and he is perfectly satisfied by this technique, which I believe he is using as a routine approach for all his mitral valve surgery.

Operations on the aortic valve were also performed using pump-oxygenator procedures. The type of pump oxygenator used by Bailey is the bubble type of oxygenator, where oxygen is bubbled through the blood while simultaneously the blood passes over certain baffles or discs, so as to increase the oxygenation, and the blood, after being filtered, passes through the special pumps into the patient's circulation.

I feel at this stage that a word should be said about the principles underlying the pump oxygenator apparatus, as many of you will not be acquainted with this very specialized type of work. Catheters are inserted into the superior and inferior vena cavae through the right atrial chamber. These catheters drain most of the blood returning to the heart. This blood is then passed through the pump oxygenator system, and returns through a single catheter introduced into the left sub-clavian artery. As a result of this by-pass, the heart is relatively dry. The term 'relatively' is correct, because blood will still flow through the coronary vessels into the heart, and thence back into the right atrium through the coronary sinus. This blood must be aspirated away even though the by-pass system is in operation. The pressure of blood in the aorta keeps the aortic valves closed, so that the blood can circulate through the systemic system without actually circulating through the heart, except for that blood which passes through the coronaries, and the blood which reaches the heart through the bronchial system. This means that the bronchial arteries coming off the aorta will return blood through the pulmonary veins, which enter the left auricle, thus coming into the field of operation. This blood must also be aspirated away during the by-pass procedure.

At Bailey's Clinic I saw for the first time operations for the repair of ventricular septal defects and for the repair or correction of aortic stenosis using open heart surgery. The first point that comes to mind is the question of the nourishment of the heart. Bailey makes use of retrograde perfusion of the heart muscle through a coronary sinus catheter. The aortic valve is approached through the aorta under direct vision, and repaired.

The correction of transposition of the great vessels of the heart is also performed by means of this by-pass method. At this same clinic I witnessed the Vineburg operation, devised to improve the blood supply of the myocardium in coronary artery disease. I shall describe this procedure later, as I was fortunate enough to see much of the surgical experimental work in connection with coronary disease in New York.

One of the few pre-operative investigations that I was able to see was the examination of both the right and left ventricles, using opaque dye. This is carried out as an out-patient procedure in the X-ray Department of the Hahneman Hospital. The patient is lightly sedated, and under local anaesthesia a needle is introduced through the skin in the region of the xiphisternum in an upward direction. It is passed upwards and outwards through the right venticle, and the dye which will outline the ventricle is then injected. The needle as it is advanced will pass through the septum into the left ventricle, which can then be outlined with the dye, which will then pass up into the aorta. If mitral incompetence is present the dye will then pass into the left auricle. Great care must be taken to ensure that no dye is injected into the ventricular septum, as this will result in a permanent heart block, and may even cause the death of the patient.

This examination of ventriculography is believed to be one of the only reliable methods of determining the presence and degree of mitral and tricuspid incompetence. Pictures are exposed rapidly by means of a special cassette changer, and the degree of contrast shown by the dye was better than anything I have seen. The reason for this may be partly due to the fact that 90% hypaque was used. This opaque medium is not at present available in South Africa.

LILLEHEI AND VARCO, MINNEAPOLIS

From Philadelphia to Minneapolis, to see the work of Lillehei and Varco, who — with DeWaal — had developed the pump oxygenator apparatus. A few words first about the surgical department at the University of Minnesota in Minneapolis, under the control of Dr. Wangensteen. A great deal of the renown of this school of medicine is due to the personality and drive of this great surgeon. Many surgeons will have heard of Dr. Wangensteen's views on carcinoma of the colon, and the 'second look' operation which he has advocated. This second look operation is usually done about 6 to 9 months after the first operation, and patients—irrespective of their financial status—return to the hospital for a re-examination operation carried out at the expense of the hospital. A great deal of information about the spread of cancer of the colon has been gleaned, as well as knowledge of the efficacy of modern surgery.

A special type of operation is practised in Dr. Wangensteen's department for the treatment of malignant disease of the breast. The surgical pathology department found that the glands along the internal mammary artery were involved in a large number of cases in whom the carcinoma appeared to be localized to one quadrant of the breast. Consequently a super-radical mastectomy is now performed, which is really 'super' and 'radical'. An ordinary radical removal of the breast and axillary lymphatics is followed by dissection of the supraclavicular glands and a sternum-splitting

operation to remove the glands of the internal mammary artery inside the pleural cavity. This extensive operation is, in Dr. Wangensteen's opinion, justified, because almost 30% of cases show the presence of involvement of these regional lymphatics.

The University of Minnesota, situated in Minneapolis, is in one of the wealthiest areas of the United States. Minnesota is, in fact, referred to as 'the bread-basket of the nation'. Dr. Wangensteen has collected large sums of money for the research work carried out by his Department. The operating theatres, which are not very large, are certainly some of the most magnificent I have ever seen. Visitors are shown to a room immediately above the theatre, in the floor of which is a dome-shaped projection made of thick glass. The visitor is thus able to sit in comfort, placed immediately over the operating table, and is given opera glasses to ensure a close-up view of what is going on in the theatre below. I was told that at one time a two-way inter-communication system existed, which was discontinued because of some of the adverse comments heard by the operating surgeons from a group of visiting surgical authorities. I cannot vouch for the truth of this, but I am sure that a two-way inter-communication system would be extremely distracting to the surgeon.

This University Hospital has an impressive heart hospital attached to it, with at least fifty beds for surgical cases. It has been donated entirely by the efforts of the Variety Performers Guild, and houses the patients on whom operations have been performed. There is communication with the main hospital by a most complicated system of passages, and interestingly enough, at many vantage points in the passages, there are special machines, from which one can buy hot coffee or tea, with or without milk, and with or without sugar, according to one's taste.

Drs. Lillehei, Varco and DeWaal have their names associated with a special type of pump oxygenator, and it will be recalled that they were the first group of workers to use human donors. The mother or father of a child suffering from Fallot's Tetralogy would act as a donor supplying the blood and the oxygenation system for the child patient. I was told that no parent was ever lost, but some cerebral damage had been suffered by one of them. The development of an artificial oxygenator and pump was inevitable, and Drs. DeWaal, Lillehei and Varco then perfected the pump oxygenator, which is used in many parts of the States. The pump oxygenator is a Sigmamotor pump, which consists of various finger-like projections, which massage the tubing in which the blood circulates. This machine gives the blood projectile force resembling the stream issuing from an ordinary heart, i.e. there is a systolic and diastolic ejection from the machine. The blood, however, which comes from the patient must also pass through an oxygenator. The type of oxygenator used in this department is the bubble type, i.e. blood is collected in a tube, and oxygen is bubbled through it. The blood then overflows into a reservoir or de-foaming chamber, following on which it is collected in a special coiled tube called the Helix, and having passed through filters, is pumped into the patient.

A team of workers is necessary to work this type of pump oxygenator. This team consists essentially of three people—a technician and two doctors, one of whom must be trained to regulate the blood flow. The apparatus is assembled in the theatre just before the commencement of an operation, and one must remember of course that the machine itself must be primed with blood, and this may require as much as five pints per patient. Also the blood must be heparinised immediately before use. Heparinised blood cannot be kept longer than an hour or two at the most, and it would be extremely dangerous to use it if it had been left standing for several hours.

The assembling of five pints of blood may require the testing of 50 or 60 donors before the correct blood is found, and careful attention must be paid to the cross-matching and compatibility of the blood for each patient. It can thus be seen that the actual preparation of the apparatus for use involves considerable expense.

Many cases with a pure ventricular septal defect have shown evidence of pulmonary hypertension, and Drs. Lillehei and Varco believe that it is this pulmonary hypertension which has been responsible for some of the post-operative fatalities. Secretions in the bronchial tree may become uncontrollable, so they perform a routine tracheotomy on their patients a week or so before the operation.

I saw the pump oxygenator in use for cases of ventricular septal defect, cases of Fallot's Tetralogy, cases of aortic aneurysm resection, and also for transposition of large vessels. Its actual use at the time of operation is very much the same in all cases. At this hospital the operations were done by two senior surgeons with two assistant surgeons. Two junior surgeons stood by, and they were washed-up merely in order to get acquainted with the nature of the operation. The pump oxygenator was connected to the patient firstly through the vena cavae, the venous blood was drained into a sump, which was then pumped through the pump oxygenator system, and returned to the subclavian artery.

Some of the cases of Fallot's Tetralogy which I saw treated required not only a plastic repair of the ventricular septal defect, but also a plastic widening of a hypoplastic pulmonary artery. This requires a small insertion of Ivalon into the myocardium in the region of the pulmonary artery so as to increase its size.

The surgery that I saw in Minneapolis was of a very high standard. I was able to watch a number of the cases in the immediate post-operative period. For the first few days the patients were kept in a resuscitation ward, which in itself was extremely interesting. As one would expect, not all cases go according to plan—e.g. during the repair of a ventricular septal defect, the bundle of His was caught up in the sutures, as a result of which the patient developed complete heart block. The post-operative management of all cases naturally calls for continuous attention from the medical staff. At one time I saw three patients in the post-operative recovery room, each with the following equipment attached to him—a tracheotomy tube with oxygen supply, and a suction tube available in case secretions became excessive. Two drainage tubes had been inserted lower down on each side of the chest, while stitched over the front part of the chest was one electrode of a special pace-maker, the other electrode being attached to the myocardium. The patients also had catheters in position in order to collect all urine, and intravenous cannulae were in position in both the antecubital and the femoral veins. All patients require oxygen, and cases with heart-block require a pace-maker together with its mechanical signal in operation. The whole collection of patient and apparatus was placed on a special rocking bed, in order to assist respiration.

Obviously such careful post-operative care is only available in these very highly specialized centres. I must mention that careful investigations were made in all cases to try and determine the cause of the development of complete heart block. Several operations had been done while the patient's heart was actually contracting, so it was decided to try several of them under a-systole; potassium citrate is injected into the aorta and renders the heart completely immobile.

This was of particular interest to me, because there was no electrocardiographic evidence that the patient was alive, as there was no heart beat. The circulation, of course, was maintained by the pump oxygenator system, and one knew that that was still working because one could see and hear the motors, and the blood being pumped through the circuit. The electro-encephalographic readings taken from electrodes pinned into the patient's scalp provided the only evidence that the patient was alive. The special curves on the E.E.G. show very clearly whether the patient is suffering from any anoxia or not.

Magnificent research and experimental laboratories are attached to this hospital. Operations are carried out on dogs regularly, and the experimental work is carefully controlled and recorded. I also visited several of the combined clinics where cases are discussed before being sent for surgical treatment, and I was impressed to see that where cardiac catheterization or angiocardiography had been done cine records were kept, so that all the staff could see a 16 mm. cine film showing the flow of dye through the heart chambers during the process of angio-cardiography. These records were kept of all their patients, and could be repeatedly shown until the combined committee had finally come to a diagnosis as to the type of lesion.

The team of workers at Minneapolis are not yet certain whether their pump oxygenator is the final answer to the problem of artificial circulation. Unfortunately one of the complications is the cerebral damage which the patient may suffer as a result of this form of artificial circulation. They are not certain whether micro-bubbles, 'anti-foam', the temperature or the biochemical changes in the blood are the cause of cerebral damage. Undoubtedly the fact that a percentage of cases suffer cerebral damage as a result of a successful by-pass operatiom must make all workers realize that there is yet room for improvement in the pump oxygenator field.

KIRKLIN, THE MAYO CLINIC, ROCHESTER

I had learned a great deal at Minneapolis, and most regretfully had to carry on to my third stop, namely the Mayo Clinic at Rochester, which I had always wished to see. I had decided to watch Kirklin and Ellis operate, as they employed a different type of oxygenator. I have already described two types of oxygenator, namely the bubble oxygenator used by Lillehei and Varco, and the revolving disc oxygenator used in conjunction with the bubble oxygenator as employed by Bailey. At the Mayo Clinic they used the type of oxygenator which had been initially described and built by Gibbon. It consists essentially of a series of frames of stainless steel. The blood is allowed to run over these frames rather like the battery plates in an accumulator, and while the blood runs over these steel mesh-work frames, a thin layer is exposed to an atmosphere of oxygen, CO_2, and ether, so that the gas content of the blood is altered and made suitable not only for oxygenation but also for anaesthesia of the patient. The de Bakey pump is also greatly modified and is of the revolving type which causes occlusion of a rubber tube without actually producing a wave similar to that produced by a Sigmamotor. This wave of the Sigmamotor, it will be remembered, resembles the pulse pressure wave in a normal person. This whole pump oxygenator machine is a very beautifully built piece of equipment. It is calibrated, and there are electronic devices arranged so that any increase in pressure on one side or one part of the circuit automatically speeds up or slows down the pump when necessary. An electronic device on the opposite side will also regulate the amount of blood being pumped into the patient's circulation. The blood is kept at a given temperature, and there is an intermediate circuit which allows the blood to circulate and be oxygenated without actually passing through the patient.

This special pump oxygenator machine is wheeled into the operating theatre at the commencement of operation, and is prepared by the technicians. The surgeon connects it up when the by-pass assistance is required.

The Gibbon pump oxygenator machine has undergone numerous modifications and the final apparatus is a shining, chromium-plated unit. Many hundreds of thousands of dollars have been spent in its perfection and construction, and once again we see how the amazing financial resources of the Mayo Clinic have been responsible for the development of new medical instruments. I saw the machine-tool workshops, where all the mechanical apparatus is prepared in blue prints before the final construction.

The problem of the sterilization of the apparatus must immediately come to the mind of the reader. All the tubing is autoclaved, and the stainless steel screens in the plastic containers must be sterilised by immersion in sterilising solutions as well as special autoclaving. The Bubble Oxygenator of DeWaal has the advantage of being sterilizable by autoclaving, whereas the Mayo machine can only be partly autoclaved. The DeWaal Oxygenator has the disadvantage of being open to the atmosphere, while the May machine is relatively closely sealed.

Contaminating organisms can enter these machines, so that very great care must be exercised with the sterilizing before the machines are used. I was informed that Pseudomonas septicaemias have been encountered in all centres using pump oxygenators, so that the hazard is a very real one.

The surgical technique I saw at the Mayo Clinic was of an extraordinarily high standard. The cases were mainly either pure ventricular septal defects, or ventricular septal defects associated with Fallot's Tetralogy. I also witnessed the operations for several cases of mitral stenosis, and also a few cases of aortic aneurysm. My main interest, however, was in the repair of septal defects.

I was impressed by several other things at the Mayo Clinic. Firstly, an operating theatre session was held every day in the dog laboratory in the Medical Sciences Building, and operations on dogs were performed. This building housed approximately 200 dogs, as well as numerous cats, rabbits, and other animals which were used for experimental procedures. One would have anticipated both noise and smell in such a place, but neither were present owing to the magnificent air-conditioning and sound-proofing of the whole surgical area.

The recording system at the Mayo Clinic is worthy of note. During all the major heart operations continuous recording is done, outside the theatre, by technicians who record on moving photographic film. It is possible for example to have an immediate venous pressure, arterial pressure, blood oxygen examination, or E.C.G. report issued to the surgeon through a two-way intercommunication system. Furthermore there is a two-way intercommunication system between the anaesthetist and the pump team, so that if any acceleration or deceleration is required, the surgeon need not be interrupted.

The theatre staff were extremely skilful, one theatre sister doing all the work at the operating table, threading needles with the thinnest thread in an almost miraculous fashion, and also collecting sterile instruments from a trolley placed in the theatre. These instruments presumably were placed in the trolley at the beginning of the day, and removed and replaced throughout the day's work.

I left the Mayo Clinic and travelled through Chicago, where I saw the experimental plastic oxygenator being made by the Baxter Laboratories. This curtain or envelope oxygenator is disposable, and has been used in a few human cases. It has great possibilities, though at present its use is restricted by the size of the patient. The use of this apparatus is still in the experimental stage, and it is only large enough for an average sized dog, so can only be used for small children.

I was advised to see it as used by Dr. A. Bakst in New York, but instead was shown Dr. Bakst's experimental work on revascularisation of the coronary arteries. This work is based on Vineburg's original operation, and I am convinced that in the future this will have a great effect on the management of cases with coronary ischaemia. I was greatly impressed with the specimens in which good cross-vascularization had occurred after internal mammary implantation.

I had gone to the States to see open heart surgery, and had visited three major centres. My visit was not only instructive but exciting, and I was deeply impressed with the standard of the surgery that I witnessed and the kindness and hospitality that I met everywhere.

The cardiac surgeon has travelled far in his chosen field and there remain but few fields left for him to conquer in his domain. It is left to the physiologist to uncover the numerous mysteries of the circulation under pump oxygenator conditions.

A REVIEW OF NEONATAL OBSTRUCTIONS OF THE GASTRO-INTESTINAL TRACT*

E. M. SPARROW, M.B., CH.B. (CAPE), D.P.H., D.T.M.H. (RAND), F.R.C.S. (EDIN.) †

Klerksdorp

The surgery of infancy has made considerable progress during the past two decades. In particular a continuing decline has taken place in the number of infants dying from obstructive lesions. This development has taken place by virtue of (*a*) earlier recognition of serious pathological conditions, (*b*) improved supportive therapy, (*c*) prompt operative treatment and (*d*) advances in anaesthesia. Infants tolerate operative treatment remarkably well when these favourable factors are combined with careful and gentle operative technique. Infant surgery relating to obstructions arises from congenital defects. As these are frequently multiple, the necessity for careful examination to discover additional defects cannot be overstressed. Many will have no bearing on the treatment or its outcome; others may be so serious as to prevent operative interference.

In this review the sequence of lesions presented will generally

* Based on a paper read to the Potchefstroom Division of the Medical Association of South Africa.

† Formerly Surgical Registrar, Bristol Royal Hospital for Sick Children and University of Bristol.

follow their position in the gastro-intestinal tract. Operative details and techniques are not included.

THE OESOPHAGUS

Atresia

By definition, the obstruction in atresia is complete. Upwards of 90% have, in association with the discontinuity, a fistula between the lower segment and the trachea. A variety of arrangements occur in the remainder. Immediate recognition of the obstruction is important. Significant features include apparently excessive salivation owing to the blocked swallowing mechanism, hungry attempts at feeding followed by regurgitation, and gagging and coughing, possibly with cyanosis, owing to laryngeal spill-over. The latter, together with tracheal aspiration of gastric juice *via* the fistula soon causes serious pneumonia. Confirmation of the block is obtained by passing through the nose a fine soft rubber urethral catheter, which will impact at the level of obstruction. A plain X-ray of the abdomen will show a gastric air-bubble if a fistula is present. A few c.c. of lipiodol (not barium) instilled into the oesophagus via the nasal catheter will demonstrate the block.

Dangers which must be avoided are aspiration pneumonia from saliva, feeds and gastric juice, and dehydration. The infant should be propped up and the secretions aspirated *via* a nasal catheter. Penicillin should be given to prevent pneumonia. Parenteral fluid to the amount of 10 c.c. per lb. body-weight twice daily plus the amount aspirated should be administered. It may be given subcutaneously with hyalase as half-strength saline with 5% glucose water in equal amounts, or intravenously without hyalase.

Examination for other defects is made. Some of these may require operative relief, e.g. imperforate anus. Thoracotomy is conducted as soon as the baby is in fit condition. This may require a delay of up to 36 hours after the initial diagnosis is made and treatment is begun.

Congenital Stenosis

Because of the fluid nature of the diet, this lesion does not present early in life unless the stenosis approaches complete occlusion. A proportion of cases will present when the change-over to semi-solid or solid foods takes place. Characteristic early features are regurgitation at feeding time, a tendency to choke from tracheal spill-over, and failure to thrive. A thin barium-mixture swallow will show the stricture with some degree of dilatation of the oesophagus above. The sheet anchor of treatment is repeated dilatation of the stricture preceded in some instances by gastrostomy. In refractory cases resection is necessary.

LESIONS OF THE CARDIO-OESPHAGEAL AREA

Achalasia, Spasm and Stricture

Peptic oesophagitis causing spasm and symptoms of stricture sometimes occurs in this age-group. In time, spasm from inflammatory lesions will gradually merge imperceptibly with *fibrous stricture*. *Neuromuscular imbalance* will show similar features. Differentiation between congenital stenosis, neuromuscular imbalance and spasm is not of practical importance, because unrelieved spasm will require dilatation as well as achalasia and stenosis.

Chalasia and Hiatus Hernia

Chalasia refers to a neuromuscular disorder in which the lower oesophagus and stomach are lacking in normal tone and peristaltic movement. The pinchcock action at the oesophago-gastric junction is absent. The surgeon may be called to see the case because of regurgitation and vomiting of feeds soon after birth. Horizontal posture will be observed to make the condition worse. If severe, the baby will not thrive. Oesophagitis and pulmonary complications will follow.

Similar features are shown with hiatus hernia. Barium swallow will show free regurgitation in both instances, with a sliding element in addition in hiatus hernia. The latter condition may be associated with spasm and in time may lead to stricture formation. Conservative measures should include frequent smaller and perhaps thickened feeds, semi-upright posture, and antacids.

THE STOMACH

Congenital Pyloric Stenosis

About three-quarters of babies with this obstruction are males. There is a higher incidence in first-born babies and occasionally a familial incidence. Few show symptoms before the 10th day, when the child begins to regurgitate feeds, returning more after each feed until the vomit becomes forceful in character. With less entering the bowel, the child becomes progressively more constipated and fails to gain weight or loses weight and becomes dehydrated. Unless weakened by starvation, hungry attempts at feeding are made. Diagnosis in the great majority is established clinically. As the obstruction is pyloric, the vomitus does not contain bile. The time of onset rules out an atresia. Oesophageal stenosis will be seen to be unlikely because the child accommodates a good volume of the feed before returning it, and there is no coughing and gagging. The triad of the typical history, visible gastric peristalsis, and the palpation of the pyloric tumour in the right upper abdomen, establishes the diagnosis. In doubtful cases, pylorospasm, bad feeding regime, incomplete rotation of the gut, annular pancreas, and intracranial injury have to be considered. In cases which have not progressed to starvation, dehydration and electrolyte imbalance, operation is probably safe without pre-operative parenteral fluids; pre-operative oral glucose water and glucose saline will suffice. In worse cases parenteral fluids should be given. Nasogastric deflation is adopted in all cases before operation.

THE SMALL INTESTINE

Atresia and Stenosis

Atresias, as opposed to stenoses, are frequently multiple. In both cases, upwards of 95% occur in the small gut as opposed to the large gut. About half of the stenoses of the gut and a quarter of the atresias occur in the duodenum. Both are almost invariably below the level of the ampulla of Vater and the vomitus therefore contains bile. Obstructive signs are apparent soon after birth in all atresias. The majority of stenoses show up in the first month of life. The remainder present at the time of dietary change and a few during childhood—the latter often with a history of abdominal pains. Vomiting, at first of biliary material and later of brownish fluid when the obstruction is in the jejunoileal, area, is the first symptom. Distension, not present in high obstruction, is usually well marked, with 'laddering' from successive loops in lower lesions. The anus should be inspected and gentle digital rectal examination conducted with a well lubricated little finger. Farber's test—the microscopic examination of stained meconium for cornified epithelial cells—is useful. If negative, an atresia is present. If positive, stenosis or some other obstruction (excluding atresia) exists. Radiology may or may not show distended loops or fluid levels if the obstruction is high. The speckled appearance of meconium ileus is absent. In isolated cases, spotty calcification from healed pre-natal perforation and sterile peritonitis may be seen. Laparotomy must be undertaken whenever possible. Important pre-operative measures include examination for other deformities, parenteral fluid, nasogastric decompression, vitamin K, and antibiotic cover—the latter to avert chest complications.

Meconium Ileus

This generalized disease first manifests itself by intestinal obstruction caused by thick, tarry, tenacious meconium. There is widespread disturbance of the mucus-secreting glands of the respiratory and gastro-intestinal tracts, with reduction of pancreatic secretions. Relief of obstruction is followed by disturbances of nutrition and proclivity to respiratory infection. Intestinal obstruction is apparent soon after birth. There may have been one or two stools or complete constipation. The well-marked distension indicates a low bowel obstruction. Factors which favour a diagnosis of meconium ileus are: (*a*) A history of obstruction in another member of the family—occasionally more than one member is affected, (*b*) the palpation of pultaceous masses in the lower abdomen, and (*c*) wide variation on X-ray in the size of the intestinal loops and granular appearance of the putty-like masses of meconium. At times the stippling of calcification referred to above is present. Important measures in therapy include antibiotic cover, the correction of fluid and electrolyte deficit, naso-gastric suction, and the administration of vitamin K.

Blood should be available at operation as well as pancreatic enzymes for post-operative use.

Anomalies of Rotation of the Gut

The majority of cases requiring operation for malrotation present during the first month of life. Well-marked obstructive features are shown by this group and those occurring during babyhood. The smaller number presenting during childhood do so with an intermittent history.

These obstructions arise as follows: (*a*) Duodenal obstruction caused either by an incompletely rotated caecum exerting pressure on the descending duodenum or by a peritoneal fold extending across this portion of the duodenum from caecum and ascending colon to the postero-lateral abdominal wall. (*b*) Incomplete rotation is associated with varying degrees of lack of fixation of the normally fixed portions of the gut, including the second and third parts of the duodenum, duodeno-jejunal flexure, terminal ileum, and ascending colon. This laxity together with the abnormally mobile mesentery encourages rotation (which is usually clock-wise as one faces the patient) of portions or the whole of the mid-gut. This duodenal obstruction and mid-gut volvulus may occur independently or, in a high proportion of cases, may co-exist. Volvulus may produce closed-loop obstruction only, or may progress to strangulation. The area affected may vary from a small sector to the whole of the mid-gut.

Clinically, there is usually a history of normal or relatively normal bowel activity at first. There is evidence of high obstruction, with bile in the vomitus. Distension may be absent until gas accumulates in the initially collapsed loops. The baby's condition deteriorates rapidly with the onset of strangulation. In cases presenting in the first few days the picture is usually indistinguishable from atresia, well-marked stenosis, or meconium ileus. Distinction is not important; pre-operative measures are the same as for these conditions.

Annular Pancreas

This abnormality is important because of the obstruction it may produce and because of the commonly associated pancreatitis. A minority of these cases (where the lumen is markedly narrowed) present in the neonatal period. The pancreatic tissue may partly or, more frequently, wholly encircle the descending duodenum. The vomitus may or may not contain bile, depending on the relationship of the constriction to the ampulla of Vater. The symptoms may be intermittent or may be those of complete obstruction. Stenosis, atresia and volvulus enter into the differential diagnosis. A plain X-ray film may show distension of the duodenum and stomach or may not be helpful. Electrolyte and fluid imbalance should be corrected, nasogastric suction instituted, and a search made for other abnormalities before a laparotomy is undertaken.

CONGENITAL DIAPHRAGMATIC HERNIAS

Because of the complicated sectional development of the diaphragm, sites of incomplete development or of non-fusion allow of herniation. Such herniae usually do not have a sac and always extend from below upwards.

The commonest site is the left postero-lateral area of the diaphragm, where an aperture of varying size may occur. Other sites of herniation include the right postero-lateral and retrosternal areas of the diaphragm, and the oesophageal hiatus. Hernias of the oesophageal hiatus produce symptoms when reflux is marked. The oesophageal type may be of the sliding variety or, in rare cases, it may be associated with a congenitally short oesophagus (intrathoracic stomach).

Postero-lateral hernias produce circulatory and respiratory symptoms from compression of the lungs and mediastinum. Cyanosis and dyspnoea may be present from birth in severe cases or may appear during nursing or crying. Relief may result when the infant is placed on the affected side, allowing greater expansion of the opposite lung. Gastro-intestinal symptoms may be minimal, ranging from failure to thrive, with occasional regurgitation or vomiting, to severe vomiting with complete obstruction and even strangulation. Examination of the chest may show reduced movement on the affected side, mediastinal displacement and a tympanitic note on percussion, depending on the extent of the herniation. Bowel sounds may be heard in the chest. Confirmation is obtained on plain X-ray. Hiatus herniae do not exhibit physical signs.

In some hiatus herniae, and in all postero-lateral herniae, operation is indicated. Pre-operative gastric decompression is important to facilitate reduction of the viscera and closure of the diaphragm and abdomen. Cognizance should be taken of other abnormalities and fluid losses corrected. Blood should be available.

OTHER LESIONS

Internal and External Herniae, etc.

On rare occasions internal herniae—paraduodenal, retrocoecal and fenestrations of the mesentery—may obstruct. Congenital bands, such as Meckel's, or unusual peritoneal folds, may also cause obstruction and strangulation. An inguinal hernia may occasionally incarcerate in the neonatal period and when not reducible by taxis operation is proceeded with forthwith.

Atresia and Stenosis of the Colon

The colon is uncommonly involved in atresia or stenosis and when these lesions in the small bowel are multiple, the colon is spared. Distension is a well-pronounced feature. Radiological signs of obstruction are well shown. Anal inspection and rectal examination are mandatory.

Ano-Rectal Malformations

These abnormalities may be classified into the following clinical and practical groups: (1) The anus is perforate. Stenosis is present in the lower rectum or anus. The anatomy is otherwise normal but for an occasionally associated fistula. (2) Imperforate anus is present owing to a diaphragm obstruction only. This represents the least degree of imperforate anus. As with the stenosis group, fistula is only occasionally associated. (3) Imperforate anus, the rectum ending blindly at a variable distance away from the anal skin. The great majority of fistulae are associated with this group. Usually the anal musculature is present and a dimple represents the anal site. The majority of ano-rectal malformations fall into this group. (4) The lower rectum and the anus are normal, the upper blind rectal pouch ending at a variable distance above. Fistulae do not occur with this group, or very rarely.

About 70% of these disorders are associated with fistulae of one type or another. In males, fistulae communicate with the trigonal area of the bladder (recto-vesical), with the membrano-prostatic urethra (recto-urethral), or with the perineum anterior to the anal area. In females they communicate with the vagina (recto-vaginal), with the fossa navicularis posterior to the hymen, or with the perineal skin between anus and vagina.

Only about half the fistulae seen are wide enough to allow evacuation of the rectum. When they are large the defects may remain unrecognized for some time. The great majority of fistulae are associated with an imperforate anus with the rectum ending at a variable distance above. Careful examination of the perineum is conducted and, in males, urine examination for faecaluria is carried out. External and approachable vaginal fistulae are probed in order to determine the lower limit of the rectum, and palpated externally with a finger. Where an open anus exists, digital examination to determine a possible rectal block (group 4) or a bulging anal diaphragm (group 2) is carried out. Other abnormalities are searched for.

In imperforate cases a plain X-ray is of great value. By the end of the first day of life, gas reaches the farthest extent of the rectum and at this stage an X-ray (preferably lateral view) is taken, with the baby inverted and with a marker at the anal dimple. The distance from the blind pouch to skin can thus be gauged. This information (helped by that obtained by probe where possible) is of great practical assistance in determining the approach to be used in group-3 cases. Immediate operation is essential in the obstructive lesions. The type of surgery conducted is dependent on a number of factors, including the group into which the lesion falls, the presence of a fistula and its type, the degree of obstruction, the distance of the blind pouch from skin, the co-existence of other abnormalities, and the skill of the surgeon and the anaesthetist in handling this type of case.

OFFICIAL ANNOUNCEMENT: AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES: OVERDUE ACCOUNTS

The attention of members is invited to the paragraph in the General Preamble on page 1 of the *Tariff of Fees for Approved Medical Aid Societies* which makes provision for the submission of accounts which have been three months overdue, direct to the Medical Aid Societies concerned. Medical Aid Societies have been experiencing difficulties in dealing with these accounts owing to the fact that medical practitioners do not always include details as to the business address etc., of the member to whom services were rendered.

An earnest appeal is made to all practitioners, where possible, to include the following information when submitting overdue accounts to Medical Aid Societies:

1. Full name of patient and relationship to member (where applicable).
2. Full name of member.
3. Membership number of member (where applicable).
4. Home address of member.
5. Business address of member.

The inclusion of these details will be in the best interests of medical practitioners, as it will enable the Medical Aid Societies concerned to effect a more prompt settlement of these overdue accounts. Item 5 is of great importance, especially in regard to societies with branches all over the country or with several branches in the same town, e.g. the Banks' Medical Aid Society. Not infrequently members with the same name (and even the same initials) are found in large societies and it is only possible to trace the member concerned if the fullest information is supplied. It is therefore respectfully suggested that practitioners instruct their receptionists as a routine measure to obtain these particulars from all Medical Aid Society patients at their first visit.

L. M. Marchand
Associate Secretary

Medical House
35 Wale Street
Cape Town
8 October 1957

MEDIESE HULPVERENIGINGS: AGTERSTALLIGE REKENINGS

Die aandag van lede word bepaal by die paragraaf in die algemene inleiding op bladsy 1 van die tariefboek vir goedgekeurde mediese hulpverenigings, waarin voorsiening gemaak word vir die direkte indiening by die betrokke mediese hulpverenigings van rekenings wat drie maande agterstallig is.

Mediese hulpverenigings het moeilikheid ondervind met hierdie rekenings omdat geneeshere nie altyd besonderhede omtrent die besigheidsadres ens. van die pasiënt aan wie dienste gelewer is, insluit nie.

'n Dringende beroep word op alle geneeshere gemaak om tog, waar moontlik, die volgende inligting te verstrek wanneer hulle agterstallige rekenings by mediese hulpverenigings indien:

1. Volle naam van pasiënt en verwantskap aan lid (indien van toepassing).
2. Volle naam van lid.
3. Lidmaatskapsnommer van lid (indien van toepassing).
4. Huisadres van lid.
5. Besigheidsadres van lid.

Die insluiting van hierdie besonderhede sal in geneeshere se eie belang wees, daar dit die betrokke mediese hulpverenigings in staat sal stel om meer stiptelik hierdie agterstallige rekenings te vereffen. Item 5 is van besondere belang, vernaamlik i.v.m. verenigings wat takke oor die hele land of verskeie takke in dieselfde stad het, byvoorbeeld die Hulpvereniging van die Banke. Dikwels gebeur dit by groot verenigings dat daar lede met dieselfde van (selfs met dieselfde voorletters) is en dit is alleen moontlik om die betrokke lid op te spoor as die volledigste informasie gegee word. Dit word dus beleef aan die hand gedoen dat geneeshere hul ontvangsdames gelas om, as roetine maatreël, die nodige besonderhede van alle pasiënte wat lede van hulpverenigings is by hul eerste besoek te kry.

L. M. Marchand
Medesekretaris

Mediese Huis
Waalstraat 35
Kaapstad
8 Oktober 1957

PASSING EVENTS: IN DIE VERBYGAAN

Dr. W. P. U. Jackson, of Cape Town, has been asked to take part in the 3rd Congress of the International Diabetes Federation in Dusseldorf in July 1958 and also the World Congress of Gastroenterology in Washington in May 1958.

* * *

Dr. Dudley W. Burton, M.B., Ch.B. (Edin.), M.R.C.P. (Edin.) has commenced practice as a Specialist Physician at the Medical Centre, 14 St. Matthews Road, East London. Telephones: consulting rooms 5310; residence 4077; if no reply 5515.

* * *

Church Service for Doctors. A special service for Doctors is to be held in St. Stephen's Church, Pinelands, on Sunday, 20 October at 7.30 p.m. A similar service has been held in the last two years and has been largely attended. The object is to provide an opportunity for those engaged in the practice of medicine to come together to unite in a service of prayer and dedication for the work in which they are engaged, and the patients with whom they are concerned, and also to provide an opportunity to many, conscious of a debt of gratitude for the benefit they have received, to give thanks and pray for those through whose skill and devotion they have received so many blessings.

* * *

Dr. Joseph Wolpe, M.D., will be returning to Johannesburg with his family early in November after an absence of 15 months overseas, to resume his practice of psychotherapy of nervous and psychosomatic states. During the year 1956-57 he continued his researches on the aetiology and therapy of neurosis under a Ford Foundation Fellowship at the Center for Advanced Study in the Behavioral Sciences, Stanford, California, USA. In the course of his travels he lectured at numerous universities and psychiatric institutions in the United States and England. Dr. Wolpe's book, *Psychotherapy by Reciprocal Inhibition* will be published by Stanford University Press early in 1958.

As before, Dr. Wolpe's consulting rooms will be at 318 Pan Africa House, Troye Street, Johannesburg, but in future he will see patients by consultation only.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van gedugte epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk, 27 September tot 3 Oktober 1957.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	1	2	–	–	3
Kaapprovinsie ..	1	–	–	–	1
Oranje-Vrystaat ..	–	–	–	–	–
Natal	1	–	–	–	1
Totaal	3	2	–	–	5

Pes, Pokkies, Tifuskoors: Geen.

REVIEWS OF BOOKS : BOEKRESENSIES

TROPHOBLASTIC GROWTHS

Trophoblastic Growths—A Clinical, Hormonal and Histopathologic Study of Hydatidiform Mole and Chorionepithelioma. By J. Smalbraak, M.D. Pp. viii + 342. 66 Plates (8 in colour) and 9 tables. 72s. Amsterdam—London—New York—Princeton: Elsevier Publishing Company. 1957. Sole Distributors for the British Commonwealth except Canada: Cleaver-Hume Press, Ltd., London.

Contents: Preface. Introduction by Dr. Emil Novak. Introduction and some historical remarks. *Part I: Hydatidiform Mole.* I. Definition of hydatidiform mole; some conceptions as to the origin of the trophoblast, the etiology and the pathogenesis. II. Incidence, symptomatology and clinical diagnosis of hydatidiform mole. III. Hormone production in molar pregnancies. IV. Histopathologic appearance of hydatidiform mole; grading of our cases of mole according to and compared with the classification of Hertig and Sheldon and of Hunt, Dockerty and Randall. V. Therapy of hydatidiform mole. *Appendix to Part I. Part II: Chorionepithelioma.* VI. Some conceptions as to the nature of chorionepithelioma reflected in the nomenclature of the last decades. VII. Definition of Chorionepithelioma; conceptions as to etiology and pathogenesis. VIII. Incidence, symptoms and diagnosis of chorionepithelioma. IX. Therapy of chorionepithelioma. *Appendix to Part II.* Description of seventeen cases of chorionepithelioma. Summary. Bibliography. Author Index. Subject Index.

This book by Dr. Smalbraak is in the nature of a thesis on hydatidiform mole and chorionepithelioma. The literature is thoroughly covered and his cases are well put forward. While it is readily understood that conflicting ideas on the subject have to be handled, it is with considerable difficulty that the author extracts some of his ideas from his own mental quagmires. His subject is dealt with under the age-old yet clear 'definition, aetiology and pathogenesis, incidence, pathology, treatment, etc.'. Much of the bulk of the book is taken up by case histories. This is a valuable work for a library, both as a reference book and for the use of any postgraduate student working on this particular subject or in allied fields.

J.T.L.

CARDIOPULMONARY PHYSIOLOGY IN MEDICINE

Clinical Cardiopulmonary Physiology. Sponsored by The American College of Chest Physicians. Editor-in-Chief: Burgess L. Gordon, M.D. Pp. viii + 759. 248 Illustrations. 32 Tables. $15.75. New York and London: Grune & Stratton, Inc. 1957.

Contents: Foreword: The Impact of Cardiopulmonary Physiology on Clinical Chest Medicine. *Burgess L. Gordon.* The Beginnings of Observations on Cardiopulmonary Physiology. *J. Arthur Myers.* Normal Pulmonary Physiology. *Harold Guyon Trimble and James Kieran.* The Bronchopulmonary Segment as a Physiologic Unit. *Chevalier L. Jackson.* An Evaluation of Dyspnea Based on the Response to Therapeutic Procedures. *Alvan L. Barach.* Methods of Examination and Testing. History, Observation and Physical Examination for Determining Pathologic Physiology. *Peter A. Theodos.* Fluoroscopy and Roentgenology. *Paul S. Friedman.* Ventilation and Lung Volumes. *Joseph F. Tomashefski and Robert J. Atwell.* The Mechanics of Breathing. *Ernst O. Attinger and Maurice S. Segal.* Bronchospirometry. *Charles M. Norris.* Pulmonary Gas Exchange. *Ross C. Kory.* Cardiac Catheterization. *Howard R. Bierman.* Impairment of the Mechanics of Respiration. Paralytic Conditions. *James L. Whittenberger and Benjamin G. Ferris, Jr.* Constrictive Chest Conditions. *Hollis E. Johnson and George R. Meneely.* Collapse of the Lung. *Andrew L. Banyai.* The Diaphragm. *Arthur M. Olsen and H. Frederic Helmholz, Jr.* Surgical Considerations in Constrictive Diseases of the Chest. *Charles P. Bailey and Charles A. R. Skowran.* Obstructive Conditions of the Respiratory Tract (Fundamental). *Howard G. Dayman.* Bronchial Obstruction, Bronchitis and Bronchiolitis (Clinical). *Albert H. Andrews, Jr.* Bronchial Asthma. *Maurice S. Segal and Ernst O. Attinger.* The Pneumoconiosis. Pulmonary Function in the Pneumoconiosis. *Harley L. Motley.* The Benign Pneumoconiosis. *Oscar A. Sander.* Disabling Pneumoconiosis. *Arthur J. Vorwald.* The Sequelae of Pulmonary Mycoses. *Alvis E. Greer.* Pulmonary Manifestations of Collagen Diseases. *Howard A. Anderson and Herman J. Moersch.* Interstitial Pneumonitis, Diffuse Interstitial Pulmonary Fibrosis, Diffuse Interstitial Fibrosing Pneumonitis, Idiopathic Fibrosis; Hamman-Rich Syndrome. *Leon H. Collins, Jr.* Sarcoidosis. *Harold L. Israel and Maurice Sones.* Tuberculosis: Destruction of the Lung and Resultant Retraction; Fibrosis: Surgical Problems. *Seymour M. Farber and Roger H. L. Wilson.* Emphysema. *Edwin Rayner Levine and Chi Kong Liu.* Surgical Treatment of Obstructive Emphysema. *David H. Waterman.* Cor Pulmonale. *Irving Mack.* Chest Manifestations of Cardiovascular Disease. *William I. Gefter and Bernard H. Pastor.* Paroxysmal Pulmonary Edema. *Aldo A. Luisada.* Acquired Valvular Heart Disease: Clinical and Hemodynamic Features. *Milton W. Anderson and Earl H. Wood.* Congestive Heart Failure and Coronary Circulatory Insufficiency. *John J. Sampson and Albert Zipser.* Treatment of Cardiovascular Emergencies. *John F. Briggs.* The Diagnosis of Congenital Malformations of the Heart: Clinical and Physiologic Observations. *Robert F. Dillon and Benjamin M. Gasul.* Impairment of Pulmonary Circulation. Primary Diseases of the Pulmonary Artery. *Arthur Grishman and Simon Dack.* Pumlonary Embolism and Infarction. *Simon Dack and Arthur Grishman.* Index.

In this volume an attempt is made to cover the whole of pulmonary and cardiovascular disease, with particular stress on recent physiological methods of investigation. The main criticism is its excessive length and verbosity, common to so many American books. It has the faults of multiple authorship, in that some chapters are poor while others are excellent and, despite the editing, there is a constant repetition of conditions and descriptions throughout the book.

Despite these criticisms, it is a reference book worth having, if only for the excellent articles contained amongst the indifferent material. The chapter on normal pulmonary physiology is particularly worthwhile, condensing in a short summary the normal values and methods of investigation in respiratory disease. The part played by intermittent positive pressure in the treatment of respiratory disease is outlined in the chapter on the evaluation of dyspnoea, based on the response to therapeutic procedures. The chapter on emphysema is particularly useful.

One-third of the book is given over to cardiovascular disease and, although it would be better placed in a text-book on cardiology, it does complete the picture and is the best section of the book. The final chapter on pulmonary artery disease is particularly worthy of study. Despite the disadvantages outlined, this book should serve a useful purpose.

V.S.

ATLAS OF CLINICAL ENDOCRINOLOGY

Atlas of Clinical Endocrinology—Including Text of Diagnosis and Treatment. By H. Lisser, A.B., M.D. and Roberto F. Escamilla, A.B., M.D. Pp. 476. 148 Illustrations, including 3 in colour. South African Price £8 0s. 6d. St. Louis: The C. V. Mosby Company. 1957.

Contents: Section I. Pituitary Gland (Hypophysis Cereb Anterior Lobe (Adenohypophysis). Hyperpituitarism (States of Overfunction) 1. Gigantism (Giantism, Preadolescent Hyperpituitarism). 2. Acromegaly (Postadolescent Hyperpituitarism). Cushings Disease. Hypopituitarism (States of Underfunction). *In Children:* 3. Hypophyseal Infantilism (Pituitary Dwarfism, Levi-Lorain Infantilism, Ateliosis, Preadolescent Anterior Pituitary Deficiency). *In Adults:* 4. Simmonds' Disease (Extreme Insufficiency of the Adenohypophysis, Hypophyseal Cachexia, Sheehan's Syndrome). 5. Pituitary Myxedema. 6. Adrenal Cortical Insufficiency Secondary to Hypopituitarism (Hypoadrenotropic Addison's Disease). 7. Eunuchoidism Secondary to Hypopituitarism (Hypogonadotropic Eunuchoidism). *Posterior Lobe.* Insufficiency. 8. Diabetes Insipidus (Pituitary Polyuria). 9. Hand-Schuller-Christian Syndrome (Craniohypophyseal Xanthomatosis) (Defects in Membranous Bone (Xanthomatous Lesions), Diabetes Insipidus, Sometimes Exophthalmos). *Other Syndromes.* 10. Nonfunctioning Pituitary Tumors, With and Without Hypopituitarism. 11. Adult Aneoplastic Hypopituitarism. 12. Frohlich's Syndrome. *Section II. Hypothalamus.* 13. Hypothalamic Obesity and Other Fat Dystrophies. 14. Cerebral Neurogenic Sexual Precocity (Including Epiphysis Cerebri [Pineal Gland] in Males Only) (Macrogenitosomia Praecox). *Section III. Thyroid Gland.* Hyperthyroidism (Exophthalmic Goiter, Thyrotoxicosis, Parry's Disease, Graves' Disease, Basedow's Disease, Flajani's Disease, and Toxic Adenomatous Goiter). 16. Progressive Exophthalmos (Malignant Exophthalmos, Hyperophthalmopathic Graves' Disease). 17. Nontoxic Goiters (Struma). 18. Thyroiditis. 19. Cancer of the Thyroid. 20. Childhood Myxedema (Congenital, Infantile and Juvenile Types). 21. Cretinic Degeneration. 22. Adult Hypothyroidism (Myxedema, Gull's Disease, 'Internal Myxedema' (Escamilla-Lisser Syndrome), Primary Hypothyroidism). *Section IV. Parathyroid Glands.* 23. Hyperparathyroidism (Including Von Recklinghausen's Disease [Osteitis Fibrosa Cystica]). 24. Hypoparathyroidism (Parathyroid Tetany). *Section V. Adrenal Glands. Adrenal Cortex.* Adrenal Cortical Hyperfunction (Hyperadrenocorticism). 25. Cushing's Disease and Cushing's Syndrome. 26. Androgenic Type (Adrenogenital Syndrome). 27. Estrogenic Type (Gynecomastia in Males). 28. Mixed Types (Including Achard-Thiers' Syndrome—Diabetes of Bearded Women). 29. Congenital Adrenal Cortical Hyperplasia (Familial Congenital Adrenal Syndrome—Female Pseudohermaphroditism, Male Adrenal Sexual Precocity). 30. Primary Aldosteronism (Conn's Disease). 31. X-ray Procedures for Visualizing Hyperplasia or Tumors of the Adrenals. Hypofunction. 32. Chronic Adrenal Cortical Insufficiency—Addison's Disease. 33. Acute Adrenal Cortical Insufficiency (Types: Adrenal Crisis; Adrenal Apoplexy; Waterhouse-Friderichsen Syndrome). *Adrenal Medulla.* Hyperfunction. 34. Adrenal Medullary Tumor (Pheochromocytoma, Chromaffin Tumor, Paraganglioma). *Section VI. Pancreatic Islets (Islets of Langerhans).* Hyperfunction. 35. Hyperinsulinism (Organic, Relative, Functional). *Section VII. Testes.* Hyperfunction. 36. Precocious Puberty in Boys. Hypofunction. 37. Male Eunuchoidism (Preadolescent Origin). 38. Eunuchism. 39. Male Climacteric. Other Syndromes. 40. Klinefelter's Syndrome (Seminiferous Tubular Hyalinization With Gynecomastia, Puberal Seminiferous Tubule Failure). 41. Male Infertility. 42. Undescended Testes (Cryptorchidism). 43. Endocrine Tumors of the Testis. 44. Male Pseudohermaphroditism (Goldberg-Maxwell Syndrome). *Section VIII. Ovotestis.* 45. True Hermaphroditism (Hermaphroditismus Verus, Intersexuality). *Section IX. Ovaries.* Hyperfunction. 46. Precocious Puberty in Girls. Hypofunction. 47. Female Eunuchoidism (Preadolescent Origin) (Ovarian Eunuchoidism). 48. Female Eunuchism. 49. Ovarian Agenesis (Gonadal Dysgenesis, Ovarian Aplasia, Turner's Syndrome, Ovarian Dwarfism. Rudimentary Ovary Syndrome). 50 Gonadal Dysgenesis With Androgenicity (Gordan-Overstreet Syndrome). 51. Polycystic Ovaries (Stein-Leventhal Syndrome). 52. The Menopause. Other Syndromes. 53. Female Infertility. 54. Endocrine Tumors of Ovary. 55. Amenorrhea—Table of Causes. *Section X.*

Miscellaneous. 56. Progeria. (Hutchinson-Gilford Syndrome). 57. Mongolism Mongoloidism, Mongolian Idiocy). 58. Laurence-Moon-Biedl Syndrome (Dystrophia Adiposogenitalis With Atypical Retinitis Pigmentosa, Mental Deficiency, and Polydactylism). 59. Achondroplastic Dwarfism (Achondroplasia, Chondrodystrophic Dwarfism, Chondrodystrophia Fetalis). 60. Morquio's Disease (Familial Osseous Dystrophy, Osteochondrodystrophia Deformans, Hereditary Osteochondrodystrophy). 61. Gargoylism (Hurler's Disease, Lipochondrodystrophy, Dysostosis Multiplex). 62. Disorders of the Epiphyses (Epiphyseal Dysgenesis, Osteochondritis Deformans Juvenilis (Legg-Perthes Disease, Osgood-Schlatter Disease, Apophysitis of Os Calcis, Scheuermann's Disease, Calvé's Disease and Others)). 63. Hyperostosis Frontalis Interna (Morgagni Syndrome, Stewart-Morel Syndrome, Metabolic Craniopathy). *Section XI. Appendix.*

This is a fine book of its type. The purely 'atlas' side of the volume presents 148 whole-plates of drawings and photographs—the total individual number of illustrations being about 5 times as many. Common and rare endocrine conditions are included, together with the usual inevitable 'no man's land' disorders, such as the obesities, mongolism, achondroplasia, gargoylism and Morquio's disease. Some historically interesting photographs are included, such as some of the world-famous giants. The reviewer is disappointed that diabetes mellitus is not included, and he cannot agree with the authors' statement that it 'does not lend itself to graphic representation'. What about the diabetic 'cushingoid' babies with their abnormal pancreas and livers, the obese bearded diabetics, the patient in diabetic coma, the cutaneous complications, the perforating ulcers, the peripheral vascular lesions, the retinopathy and many more?

The references are up to date; aldosteronism for instance is included with 1956 references. On the whole the text is also up to date, though not quite up to the standard of the photographs. Naturally the former is apt to be rather scrappy since this book is, after all, an atlas and not a text-book. The Klinefelter syndrome is rather incompletely considered; Cushing's syndrome in children receives no mention. The advice regarding undescended testes is not consistent with the best teaching—gonadotropin as a routine in all cases does not seem right.

As well as clinical photographs, procedures such as extraperitoneal pneumography are illustrated, there are a few drawings and metabolic-response charts (e.g. tests for phaeochromocytoma), and there is an appendix with growth curves, dental age charts, etc. This is certainly a book for the library, for the endocrinologist, and most particularly for the general physician and paediatrician, who will find it of great value.

W.P.U.J.

CORRESPONDENCE : BRIEWERUBRIEK

FULL-TIME MEDICAL OFFICERS AND LIFE-INSURANCE EXAMINATIONS

To the Editor: I wish to draw the attention of the profession to a practice that has sprung up amongst medical practitioners in whole-time Government or municipal service.

In some big cities in the Union it is common practice amongst some of these medical practitioners to do life-insurance examinations after their hours of employment. It is common knowledge amongst some of us that one of them earns from one assurance company alone, more than £100 per month.

The busy general practitioner refuses to do examinations during his consulting hours, and will only examine candidates for insurance by special appointment at his consulting rooms. Many general practitioners refuse on principle to examine a healthy person at his house, generally at night. Insurance agents divert work from this type of practitioner for fear the candidate for insurance may change his mind if he is not examined immediately.

These full-time Government and municipal medical practitioners seem to be available at all times after 5 p.m. Many times the agent collects 3 or 4 candidates, and then the agent accompanies the doctor from one house to another, sometimes in towns several miles apart. At two guineas per examination it pays them.

Late one afternoon when visiting a woman patient of mine I found one of these practitioners examining the husband of my patient for insurance, although this couple had been, and are still patients of mine for many years.

Many a young struggling general practitioner would be only too pleased to do this type of work as a side-line. I think it is grossly unfair to them to take away this type of work from them, to be done by whole-time Government or municipal medical practitioners with good salaries. I am certain the agents know they are full-time men.

It is incumbent also on the senior medical officers of assurance companies to satisfy themselves that examinations are not done by full-time personnel. To stop this kind of practice it should also be the duty of all general practitioners to report such men to their various departments if they have positive proof. In my part of the world it is very common practice.

I may state in conclusion that this practice is not confined to whole-time general practitioners, but extends to some whole-time specialists as well.

Jaap

6 October 1957.

CHEST PAIN AS THE ONLY SYMPTOM OF GASTRIC ULCER

To the Editor: I have read with interest the article under this title by Dr. A. Shedrow[1] in the *Journal* of 10 August. I think he rightly draws attention to the fact that gastric ulceration high up on the lesser curve, gastric ulcers in hiatus hernia, and hiatus hernia complicating any other condition, sometimes present with chest pain as the initial symptom. However, I must cross swords with Dr. Shedrow when in case 1 he states that lung tumours were considered but were eliminated by clinical examination. Lung tumours cannot be eliminated by clinical examination without an X-ray.

Dr. Shedrow is apparently unable to decide in the first case whether the patient had 'extensive fibrosis of the oesophagus' which, under diagnosis, he states was present, or, as in his terminal passage, that there was an 'ulcerative lesion' present. It is just this difficulty of differentiation that makes oesophagoscopy necessary in these cases, for in the presence of ulceration early reduction of the hernia by surgical methods is mandatory to prevent development of the fibrous stricture.

The author is also a little uncertain whether persistence of symptoms after operation for hiatus hernia is due to the presence of another lesion, as he suggests under the heading *discussion*, or to the recurrence of the hernia, as he states under *treatment*. It must be obvious that if a patient is suffering from Saint's triad with gall-stones, it is useless repairing the hernia unless at the same time treatment for the gall-stones is embarked upon. The bald statement that 'we have seen cases where surgical repair did not succeed in maintaining the hiatus hernia under control' is valueless unless we are given a statistical background stating, for example, that 4 cases recurred out of 5, or 2 out of 100.

Under case 2 I find it difficult to understand the author's statement that the patient—48 years old—had no history of digestive troubles when he states that 'food actually quietened the pain, which often came on at night'. I thought that was typical of hunger pains and certainly was a digestive symptom.

The same applies in case 4 in that Dr. Shedrow states that in recent months the chest pain had eased off but abdominal pain had made an appearance. In the next paragraph he states that there is absolutely no sign or symptom pointing to gastric disease.

In passing, it should be noted that Barrett's is spelt thus and not as in the script.

Despite the above criticisms I enjoyed reading the article for I thought it brought into focus the fact that hiatus hernia, if present, lay in the chest and therefore chest symptoms were common. In an article published by Mr. Fuller and myself[2] in the *Journal* of 27 November 1956, we stated that in 68% of our cases of hiatus hernia retrosternal pain was present.

David Adler

22 Kotze Street
Johannesburg
19 September 1957

1. Shedrow, A. (1957): S. Afr. Med. J., **31**, 802.
2. Fuller, D. and Adler, D.: *Ibid.*, **28**, 1007.